EPIDEMIOLOGICAL STUDY OF MATERNAL AND FETAL OUTCOMES IN ABRUPTIO PLACENTA

Dissertation submitted to

The Tamilnadu Dr. M.G.R. Medical University

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OBSTETRICS AND GYNAECOLOGY

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Chennai

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BONAFIDE CERTIFICATE

This is to certify that the study entitled "EPIDEMIOLOGICAL STUDY OF MATERNAL AND FETAL OUTCOMES IN ABRUPTIO PLACENTA" is a bonafide work done by Dr.V. HEMAMALINI at K.A.P.V. Government Medical College, Trichy during the period of Post Graduate study in Obstetrics and Gynaecology from May–2007 to March–2010 under the guidance of Prof.Dr.K. RUCKMANI, M.D., D.G.O. This dissertation is submitted to Tamilnadu Dr.M.G.R. Medical University in partial fulfillment of University rules and regulations for the award of M.D. degree in Obstetrics and Gynaecology.

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DECLARATION

I, Dr. V. HEMAMALINI, solemnly declare that the dissertation titled, "EPIDEMIOLOGICAL STUDY OF MATERNAL AND OUTCOMES IN ABRUPTIO PLACENTA" is a bonafide work done by me at K.A.P.V. Government Medical College, Trichy, during 2008 - 2009 under the guidance and supervision of Prof. Dr. K. RUCKMANI, M.D., DGO., Professor in department of Obstetrics and Gynaecology. This dissertation is submitted to the Tamilnadu Dr. M.G.R. Medical University, in partial fulfillment of University rules and regulations for the award of M.D. Degree (Branch – II) in Obstetrics and Gynaecology.

Place: Trichy
Date : **Dr. V. HEMAMALINI**

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CONTENTS

INTRODUCTION

Abruptio placenta is still a grave obstetric emergency contributing large number of maternal deaths in developing countries. Bleeding from the maternal reproductive tract in which the cause is unclear is dangerous to the fetus also.

Early diagnosis of abruption is very important for improving maternal and fetal outcome to great extent. Premature separation of placenta affects the fetus mostly and the perinatal mortality as a consequence of this problem is very high.

In our hospital we have 8,118 deliveries of which the incidence of abruption was 1.26%.

Abruption – Nomenclature

Definition – It is a premature separation of normally situated placenta after 28th week of pregnancy, but before birth of the child.

Other names- *abalatio placenta*

- *premature separation of the placenta.*

Edward Rigby (1774) identified accidental hemorrhage as separate entity. Lee (1848) and *Coole* (1848) used the term *placental apoplexy.* De Lee (1892) used abruption which denotes sudden accident.

Rudolf Holmer (1901) interpreted abalation placenta as *carrying away of placenta*. This term deeply routed in British literature as accidental hemorrhage. In Latin abruption means *rending asunder of placenta,* denotes a sudden accident, a clinical characteristic of most cases of this complication.

Some of the bleeding of placental abruption usually insinuates itself between the membranes and uterus, and then escapes through the cervix, causing external hemorrhage. Less often, the detached placenta and the uterus, leading to concealed hemorrhage. Placental abruption with concealed hemorrhage carries with it much greater maternal and fetal hazards, not only because of the possibility of consumptive coagulopathy, but also because the extent of the hemorrhage is not appreciated and the diagnosis typically is made later (Change and Co-workers, 2001).

HISTORY AND REVIEW OF THE LITERATURE

Abruption was first recognized by LOUIS BOURGOIS in 1609 and was differentiated by RIGBY in 1776 from placenta previa. Because of explosive and fulminant nature of abruption, LEE and COOLIE in 1848 coined the term placental apoplexy. During the first half of 18th century neither classification of etiology nor improvement of condition made such progress. Meriman and Blundell testified the efficacy of artificial rupture of membrane, in the management of the dreaded condition.

Goodell in 1875 called the attention of the mortality in patient suffering from the disease followed by Boudelough (1970). Association with proteinuria was noticed by Chantervil in 1881. Peter (1901) called it as temporary hemophilia. In 1912 Couvelaire described it as hemorrhage. Kellogy (1928) found out the disturbance intravenous coagulation in this condition. Dieckman (1938) first identified hypofibirinogenemia. Showell (1941) demonstrated the intravascular conversion of fibrinogen to fibrin. Schneider (1947) and Howell agree with thromboplastin release into circulation. In 1948 Page suggested formation of fibrin from fibrinogen due to entry of thromboplastin.

In 1949 Moloney initiated fibrinogen replacement therapy. Again in 1951 Schneider stressed the fibrin embolism. Weiner worked out clotting deficiency and stressed the significance of clot observation test in 1903. Jeffcoate in 1956 explained the place of plasma substitute. Involvement of kidney with abruption was pointed out by Truleta (1947) Sophian (1955) and Sherhan and Moore (1952).

Incidence: Wide variability is reported in incidence of abruption because diagnosis is not always as simple as definition suggests.

Incidence ranges from 1 in 60 to 1 in 250 deliveries. Reported frequency for abruption averages about 1 in 200 deliveries.

Incidence of abruptions according to various authors;

Menon and Sokki (1961) – 1 in 55

Poddar (1961) – 1 in 169

Karegard and Gennsee (1986) – 1 in 225

Ananth and Colleague (1999) – 1 in 155

In parkland hospital incidence was severe enough to kill fetus (1988 – 1999) 1 in 1,550 deliveries.

Perinatal Morbidity and Mortality

As stillbirths from other causes have decreased appreciably, those from placental abruption have become especially prominent. According

to Cunningham and Hollier (1997) 12% of stillbirths were due to abruption in Parkland Hospital. These frequencies are similar to that described by Fretts and Usher (1997) for the Royal Victoria Hospital in Montreal between 1978 and 1995. The high mortality was due in part to the strong association between placental abruption and preterm delivery. Even in those infants delivered at term, however, perinatal mortality was 25 folds higher with placental abruption.

Importantly, if the infant does survive, there may be adverse sequelae. Of the 182 survivors in the study by Abdella and associates (1984), about 15 percent were identified to have significant neurological deficits within the first year of life. Similarly, of the 39 survivors delivered between 26 and 36 weeks followed by Matsuda and co-workers (2003), about 20 percent were diagnosed with cerebral palsy compared with 1 percent of gestational age – matched controls.

Aetiology

Primary causes unknown but there are several associated conditions like anemia, hypertension etc causes abruption. Incidence increases with maternal age and parity (Pritchard and Colleague in 1991). Toohey and associates in 1995 did not find this in females greater than parity 5.

Abruption was significantly associated with hypertensive disorder complicating pregnancy. Preeclampsia increased relative risks of abruption 2.1 to 4 folds. Chronic hypertension increased relative risks of abruption 1.8 to 3 folds. Abruption was severe enough to kill the fetus in half of the cases associated with hypertension (Pritchard and Co worker 1991). According to Ananth and associates (1999) there is 3 fold increased risk of abruption with chronic hypertension and 4 fold increased risk with severe preeclampsia.

Increased incidence of abruption occurs with preterm premature rupture of membrane (PPROM) and its relative risk is 2.4 to 3 folds. Ananth and colleague (1996) reported 3 fold increased risk of abruption with premature rupture of membrane. Cigarette smoking increases the risk of abruption and relative risk is 1.4 to 1.9 folds. According to Ananth and Misra (1999) there is 2 folds increased risk in smokers. Cocaine abuse is also associated with abruption as per Bingol and associates (1987).

Thrombophilia (inherited and acquired) had been associated with abruption. These include mutation genes for factor V – Leiden, prothrombin, protein C and S, methyl tetra hydro folate reductase and anti-thrombin III. Acquired antiphospholipids antibodies (Lupus anti-

coagulant) are associated with abruption (Gherman and Goodwin in 2000).

External trauma was associated with abruption in Parkland hospital (3 out of 207 cases).Kettle (1988) and Stafford (1988) also stressed the same relation. Uterine myomas especially if located behind the placental implantation predispose to abruption. Rice and associates (1989) reported 8 females with retroplacental myomas had abruption.

Pritchard and co worker identified recurrence of severe abruption in 18 pregnancies. Hibbard and Jeffcoate (1966) contended that folic acid deficiency played an etiological role in abruption. They showed 97.5% of females presenting with abruption had folic acid deficiency. Menon (1961) and Pritchard (1969) and Ray (1999) found no evidence to support this.

Pritchard (1991) observed in two cases sudden decompression of hydramnios causing abruption in a twin delivery. Decompression following delivery of first fetus may lead to pre-mature separation of placenta that endanger second fetus.

Nesbit (1958) produced experimental abruption by obstructing IVC; Crawford et al (1960) observed that abruption can occur as sequele to spinal anesthesia for deliveries.

Pathology

Pritchard (1970) extensively reviewed the genesis of placental abruption. The process was initiated by effusion of blood into decidua basalis due to pathology in uterine vessel or placenta. A decidual hematoma from uterine vessel leads to gradual separation, compression and ultimate destruction of the function of adjacent placenta.

The diapedesis of blood into myometrium acts like an ecbolic agent and is associated with uterine contraction which is localized or diffuse or tetanic. If relaxation is not taking place, uteroplacental circulation will be affected and lead to fetal hypoxia, acidosis and death.

In *uteroplacental apoplexy* (the term coined by COUVELAIRE), there is extravasations of blood into uterine musculature beneath its serosa, into broad ligaments, ovaries and sometimes intraperitonealy.

Couvelaire uterus

In the early stage, there may be no clinical symptoms. The condition is discovered only on examination of the freshly delivered placenta, which has a circumscribed depression measuring a few centimeters in diameter on its maternal surface, and is covered by dark, clotted blood.

Insome instances, a decidual spiral artery ruptures to cause a retroplacental hematoma, which as it expands disrupts more vessels to separate more placenta. The area of separation rapidly becomes more extensive and reaches the margin of the placenta. Because the uterus is still distended by the products of conception, it is unable to contract sufficiently to compress the torn vessels that supply the placental site. The escaping blood may dissect the membranes from the uterine wall and eventually appear externally or may be completely retained within the uterus.

Menon's series in 1961 abruption occurs 66% of cases prior to 36 weeks. Karegard (1986) also reported 50.8% of cases developed abruption before the end of 37^{th} weeks.

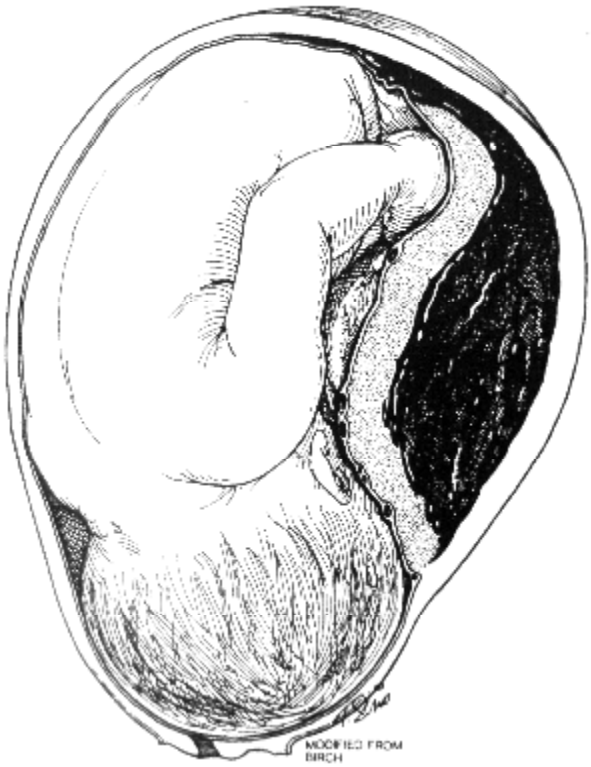
Concealed hemorrhage

Retained or concealed hemorrhage is likely when:

1. There is an effusion of blood behind the placenta but its margins still remain adherent.

2. The placenta is completely separated yet the membranes retain their attachment to the uterine wall.

3. Blood gains access to the amniotic cavity after breaking through the membranes



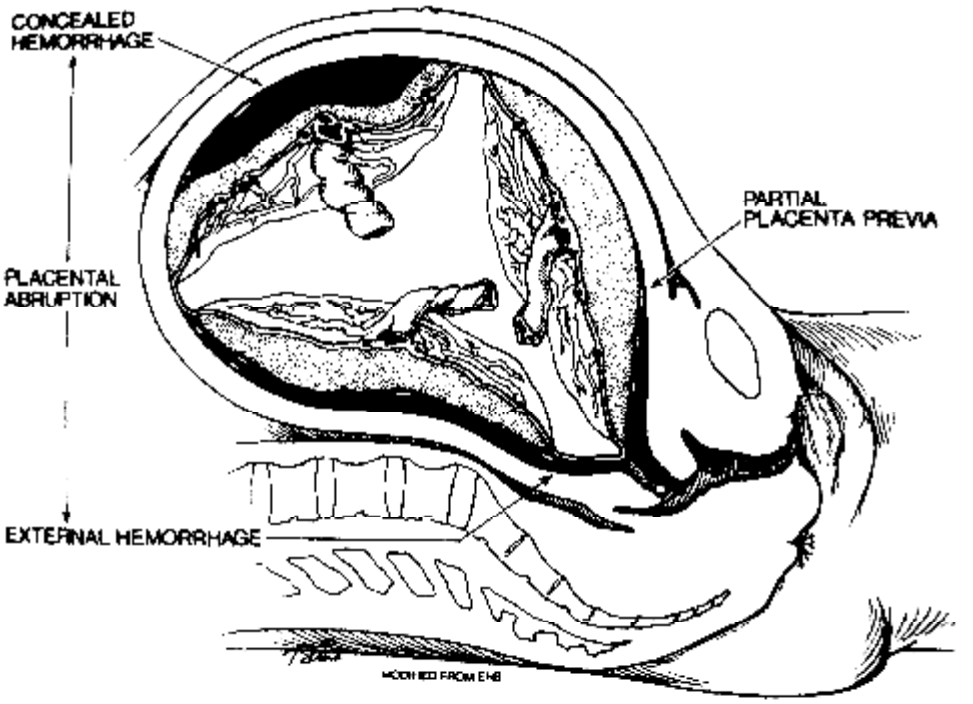
4. The fetal head is so closely applied to the lower uterine segment that the blood cannot make its way past it.

Imaging in abruptio placenta

Despite improved resolution of sonography machine, ultrasound has poor sensitivity in diagnosing placental abruption and findings are negative in most cases. Ultrasonography images correlated with the diagnosis of abruption after delivery shows a wide spectrum of images. Initially the hemorrhage may be seen as hyperechoic or isoechoic in comparison of the placenta, but as hematoma starts resolving, it becomes hypoechoic after a week.

Diagnosis

Abruption still remains on clinical diagnosis. The classical presentation seldom causes diagnostic doubt. Additional diagnostic parameters would be useful in cases with few symptoms. Diagnosis is confirmed after delivery by the presence of retroplacental clots, indenting placental substance. In these cases Sholl (1987) visualized retroplacental cleft, retromembranous clot and rounded placental contour sonographically.



However he could confirm the diagnosis in only 25% of cases with a sensitivity of 52%. Hard (1983) could recognize retroplacental hematoma sonographically in 1 out of 59 cases.

Malcus et al. (1992) studied Doppler blood flow changes in cases presented with 3rd trimester hemorrhage. They observed that there was an increased risk of abruption if arcuate or umbilical artery flow velocity waveforms are abnormal.

Differential Diagnosis

With severe placental abruption the diagnosis is generally obvious. Milder and more common forms of abruption are difficult to recognize

with certainty and the diagnosis is often made by exclusion. Unfortunately neither laboratory test nor diagnostic methods are available to detect lesser degrees of placental separation accurately.

1. Placenta praevia
2. Ruptured uterus
3. Retroperitoneal hematoma
4. Hematoma of rectus abdominis
5. Acute hydramnios
6. Non-obstetric acute abdominal conditions
7. Acute pyelonephritis

Maternal and fetal outcomes

In the recent pregnancy related mortality surveillance statistics for 1991 to 1999 published by CDC (USA) hemorrhages accounted for 17% of maternal deaths in the United States, of which placental causes have significant contribution. The proportion of maternal deaths due to hemorrhages is even higher in developing countries due to poor access to emergency obstetric care.

Perinatal Outcome

Perinatal mortality is reported to be 119 per 1000 birth in pregnancies with abruption. The adjusted relative risk for still birth is 8.9

fold as compared to singletons without abruption. Risk factors for perinatal death in pregnancies with placental abruption are smoking, severe pre-eclampsia and small for gestational age. The major causes for fetal morbidity are prematurity, fetal growth restriction, respiratory distress syndrome, anemia and hyperbilirubinemia. Extent of placental separation has a profound effect on still birth. However the risk of pre-term delivery was substantially increased even with mild abruption in surviving infant after severe abruption. Long term neurological sequelae like cerebral palsy may be four times greater.

Management

At present, opinion as obstetric management ranges from traditional conservative approach with resultant lower rate of caesarian section and high perinatal mortality to a more aggressive approach of an immediate delivery in all but mildest cases of abruption and lower perinatal mortality.

Menon et al (1961) advocated conservative management with artificial rupture of membrane was performed and labour induced or augmented by oxytocin. Caesarian section was done only if there was unfavourable response to induction. Hibbard et al (1966) also followed

the same management. Perinatal mortality in above studies was 76% and 50.5%.

Patterson (1979) adopted a conservative approach only in mild cases and in cases where the fetus had little chance of extra uterine survival. If fetus of 34 weeks of gestational age or more and vaginal delivery was not imminent, then C-section was performed with perinatal mortality of 35%.

William and Sholl advocated selective management of abruption with frequent fetal heart rate monitoring and tocolysis was advocated to promote prolongation of pregnancy and this minimized perinatal mortality from 18% to 17%.

Complications

Complications may be immediate or delayed.

Immediate

Shock

Circulatory collapse due to hemorrhagic coagulation failure

Renal failure

Atonic PPH

Coagulopathy

Delayed

Fluids and electrolytes imbalance

Anemia

Sheehan's syndrome

Puerperal sepsis

Thromboembolism

Suppression of lactation.

Shock

Intensity of shock is seldom out of proportion to maternal blood loss. Neither hypotension nor anemia is obligatory in cases of concealed hemorrhage even when acute hemorrhage has achieved considerable magnitude. Oliguria caused by inadequate renal perfusion but responsive to vigorous treatment of hypovolemia may also be observed in these circumstances. Thromboplastin from decidua and placenta enter the maternal circulation and incited intra-vasular coagulation and other features of amniotic fluid embolision.

Coagulation failure

Coagulation failure is one of the gravest complications which was first noticed by DeLEE in early 1902. DIEKMONN (1936) called

attention to hypofibirinogenemia and this was later confirmed by Weener et al. (1950).

SCHNEIDER (1955) reviewed the mechanism of coagulation defect. The explanation being entry of thromboplastin liberated by placental separation into maternal circulation through disrupted channels causing intravenous coagulation by consumption of coagulation factors, platelets, factors V, VII and fibrinogen. Small fibrin emboli may then detached and lodge in brain, liver, lungs, kidneys and micro emboli with the production of fibrin degradation products which further alter the coagulation mechanism by slowing the rate of conversion from fibrinogen to fibrin by thrombin. Hemorrhage occurs not only from uterus but also from incision, injection sites, nose, mouth and anus.

Overt hypofibrinogenemia - serum fibrinogen < 150 mg / dl with increased level of FDP > 100 μgm / ml and ‘D’ dimer are seen in abruption. Other coagulation factors are decreased in about 30% of females with abruption.

The accurate incidence of coagulation defect is difficult to record. Menon (1961) reported that coagulation failure was found in 24.8% of 125 cases. Coagulation defects are not found in mild cases. Risk of coagulation is related to degree of placental separation, amount of retro

placental clots and abruption delivery interval > 8 hrs. Available incidence indicates that deliveries are ideal often followed by treatment of coagulation failure.

Renal failure

Acute renal failure that persists in any length of time is seen in severe form of abruption. This includes in whom the treatment of hypovolemia is delayed or incomplete. Correct incidence is difficult to access due to lack of uniformity in definition. According to Pritchard and Brekka (1967) the incidence was 4%, Menon (1961) incidence was 5.5% and Eastman incidence was 1%.

Serious impairment of renal perfusion is the consequence of massive hemorrhage because preeclampsia frequently coexists with placental abruption. Renal vasospasm is likely intensified.

Atonic PPH

Uterine atony is the inability of the myometrium to adequately contract following delivery causing continuous and severe bleeding from the placental implantation site. The diagnosis is usually made by observation that the uterus it soft and boggy, contracts following massage and relaxes again resulting in more bleeding. Because of extravasation of blood into the myometrium, there may be insufficient uterine contraction.

In the last decade conservative surgical procedures have been successfully used in various circumstances (International Journal of obstetrics and gynaecology 2005)

Fluid and Electrolyte imbalance

Due to circulatory collapse and renal failure, there is electrolyte imbalance.

Anemia

Because of bleeding per vaginum and the consumptive coagulopathy there occurs anemia.

Sheehan's syndrome

Some cases manage to survive a severe and prolonged period of shock develop into sheehan's syndrome and demonstrate the signs of anterior pituitary necrosis namely amenorrhoea, genital atrophy, intolerance to cold, listerness and premature senility.

Puerperal sepsis

This is more prevalent in developing countries due to nosocomial infection, particularly operative delivery, antibiotic resistance. There is significant number of cases of puerperal sepsis due to abruption. This may be due to anemia, prolonged catheterisation.

Thromboembolism

Prolonged immobilisation leads to thromboembolism. There is also increased risk of amniotic fluid embolism due to release of tissue thromboplastin. Women with prior idiopathic venous thrombosis or positive family history of thrombosis have a high risk (>10%) and warrant active antepartum and postpartum heparin prophylaxis.

Clinical presentation from Hurd and associates (1983)

S. No.	Signs and Symptoms	Frequency
1.	Vaginal bleeding	78%
2.	Uterine tenderness / back pain	66%
3.	Fetal distress	60%
4.	High frequency contraction	17%
5.	Hypertonic	17%
6.	Idiopathic DIC	22%
7.	Dead fetus	15%

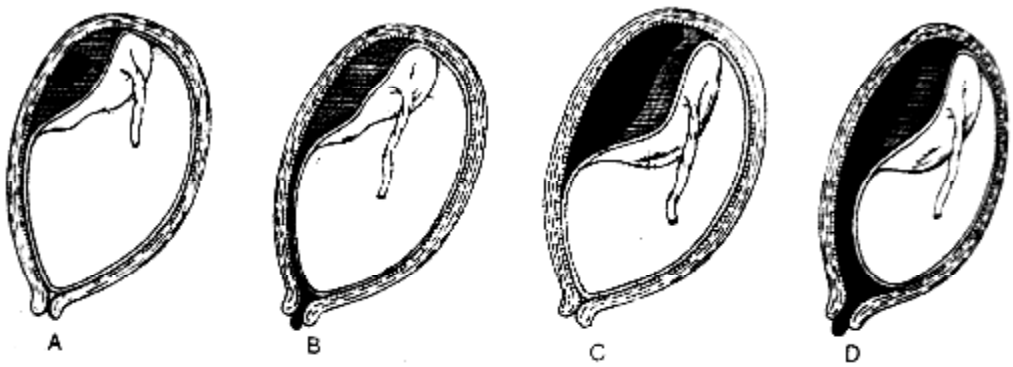
Grading in abruption

Grade – 0: Asymptomatic usually diagnosed after delivery by the presence of retroplacental clots.

Grade – 1: Mild vaginal bleeding, uterus hypertonic and tenderness present, no signs of maternal shock or fetal distress.

Grade – 2: Vaginal bleeding may or may not present. Uterine tenderness and tetany are present. No signs of maternal shock. Signs of fetal distress present.

Grade – 3: Vaginal bleeding may or may not present. Marked uterine tetany, persistence of abdominal pain and maternal shock are seen. Fetus is usually dead. Coagulopathy are seen in 30% of cases.



Grading in abruption

Maternal prognosis depends on the degree of placental separation as evidenced by maternal shock, uterine tetany and absence of fetal heart sounds, Overall blood loss, presence of complications like coagulopathy, renal failure, degree of uteroplacental apoplexy and associated vascular

diseases. Pattern of the bleeding whether revealed or concealed, abruption delivery interval and availabilities of facilities also favors prognosis.

Most of the immediate deaths are due to hemorrhage and delayed deaths are due to coagulation failure and renal failure. Almost all deaths had occurred in Grade 2 and 3 degrees of abruption. Higher degrees of suspicion, early diagnosis and definitive treatment should reduce maternal mortality within 0.5 to 5%. The tragedies of abruption exemplified by the facts that in severe cases fetal wastages was approached 100%. In mild cases fetal wastage approached 20 to 50%.

Fetal prognosis depends upon the time at which premature separation occurs, severity and degree of separation and treatment adopted. Naidu et al. reported perinatal mortality rate in Grade 2 and Grade 3 as 60-80%. Severe degree kills the fetus almost immediately and at times caesarian section in early case may help to save the fetus.

Menon (1961) reported perinatal mortality in Grade 1-31.6%, Grade 2 and 3-87%. Crohn and associate (1987) reported perinatal mortality rate was 20%. Ananth and co workers (1999) reported perinatal mortality to be about 40%. According to Theiba-B and associates (2003) maternal mortality rate due to abruption was 3% (mainly due to anemia – 61.6% and Puerperal infection – 7.9%) and perinatal mortality 8.59%.

AIM OF THE STUDY

1. To conduct an in depth analysis of abruption in order to find out the influence of various parameters.
2. To find out the incidence of abruption
3. To find out the relationship of incidence to age and parity.
4. To find out maternal and perinatal mortality rate
5. To find out various maternal morbidity parameters and their incidence.
6. To find out the factors contributing to maternal and perinatal morbidity and mortality.

MATERIALS AND METHODS USED

This is a prospective study conducted in Annal Gandhi Memorial Govt Hospital, Trichy. During the period of 2008 to 2009.

Inclusion criteria

All patients are diagnosed mainly on clinical signs and symptoms / ultrasound.

Exclusion criteria

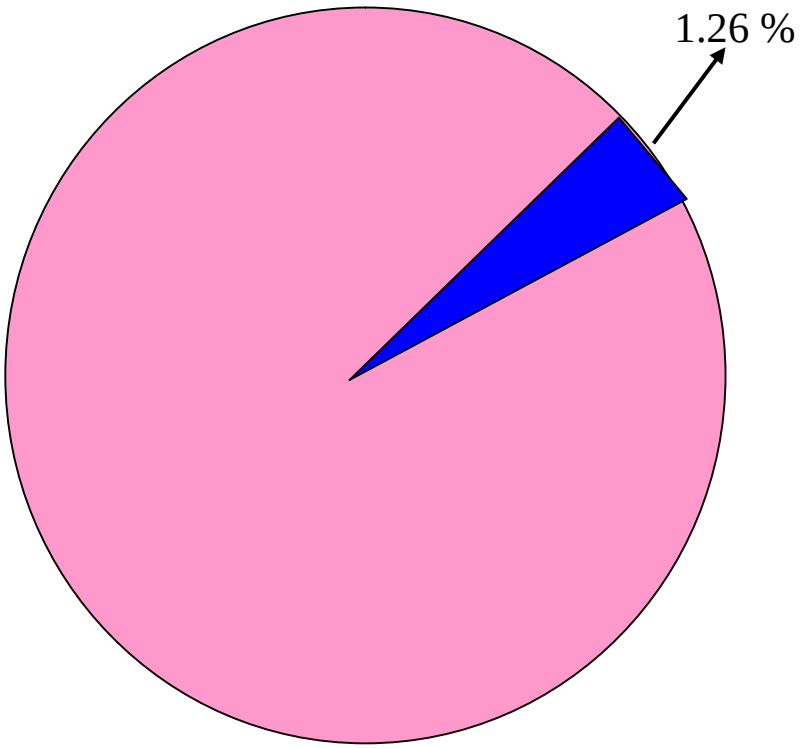
1. All patient who came with the history of Ante partum hemorrhage but where diagnosed as having local causes were excluded.
2. All patients referred to the hospital for the management of post partum complication were excluded from this study

Methods

1. Careful elucidation of history from the patients.
2. Meticulous clinical assessment so as to exclude from placenta previa.
3. Ultrasonogram done for placental localization and for retroplacental clots.

4. Venous sample taken for blood group and Rh type, cross matching, hemoglobin estimation, estimation of serum fibrinogen and clot observation test. Estimation of renal function test and liver function test whenever indicated.
5. Vital signs should be monitored regularly
6. Clot observation test repeated hourly
7. Blood transfusion and fluid infusion monitored closely.
8. Coagulation failure treated with fresh blood transfusion and fresh frozen plasma.
9. Appropriate management of renal failure when it occurs.
10. The aim of management of abruption was to expedite the delivery so as to shorten the abruption delivery interval. Liberal caesarian section was done.

Incidence of abruption



RESULTS AND OBSERVATION

This study deals with maternal and fetal outcome in abruption

Period of study

One year

From May – 2008 to April - 2009

Total Deliveries during this period

Total number of deliveries - 8,118.

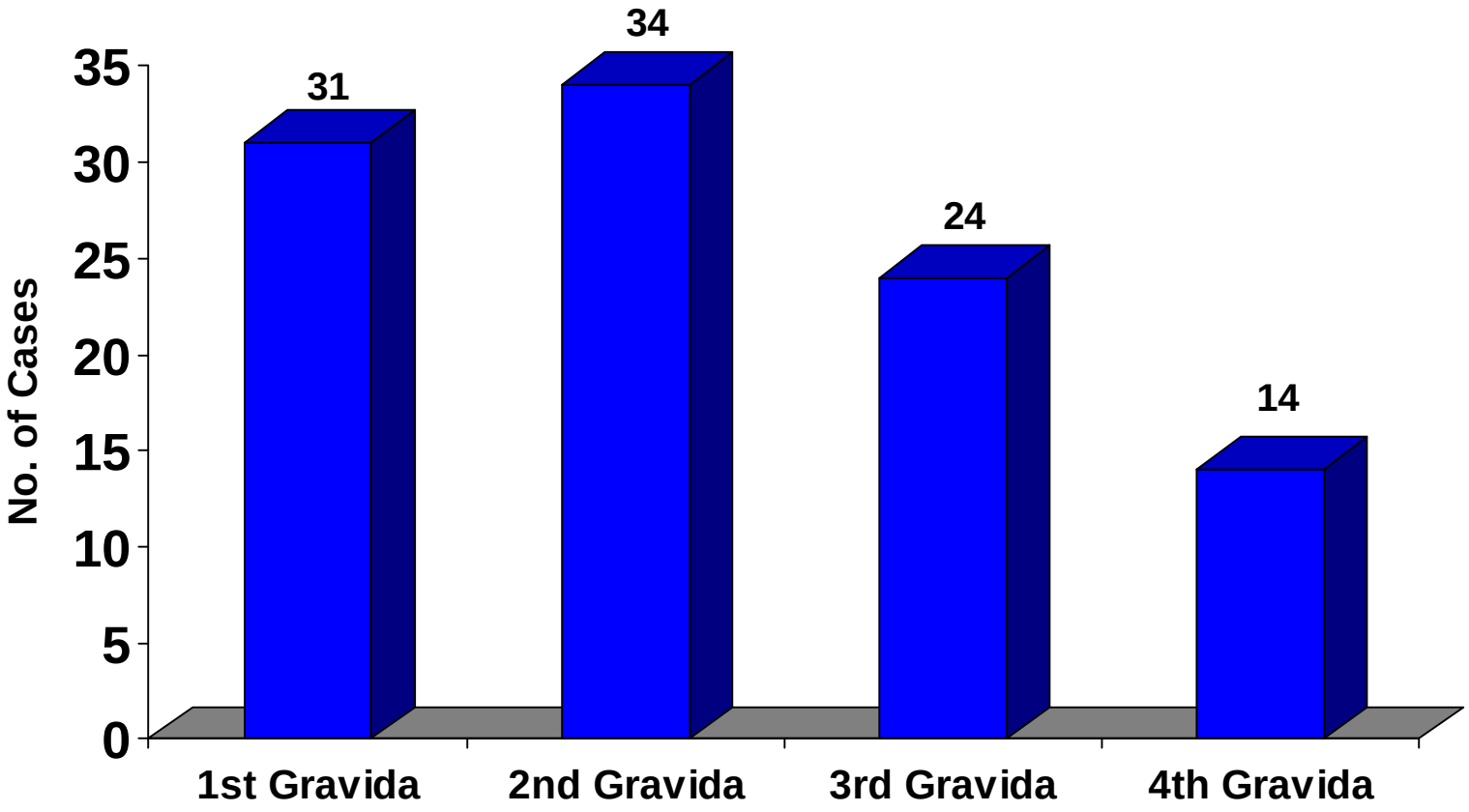
Out of which there were 103 cases of abruption.

This gives incidence of 1.26 %

Total no. of deliveries	Total no. of abruption	Incidence
8,118	103	1. 26%

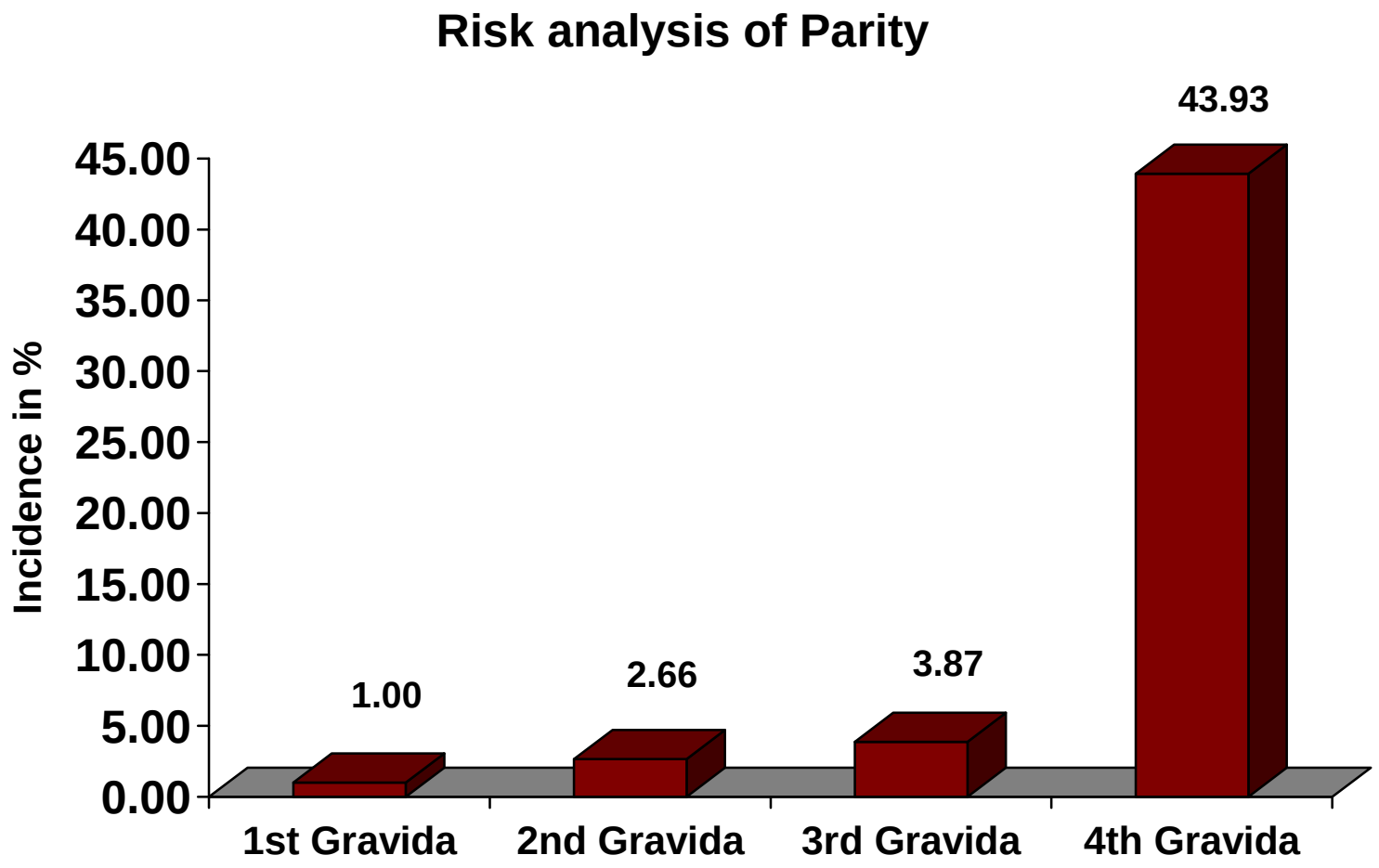
Incidence: 1 out of 79 cases.

Relationship between Parity & Number of cases of abruption :



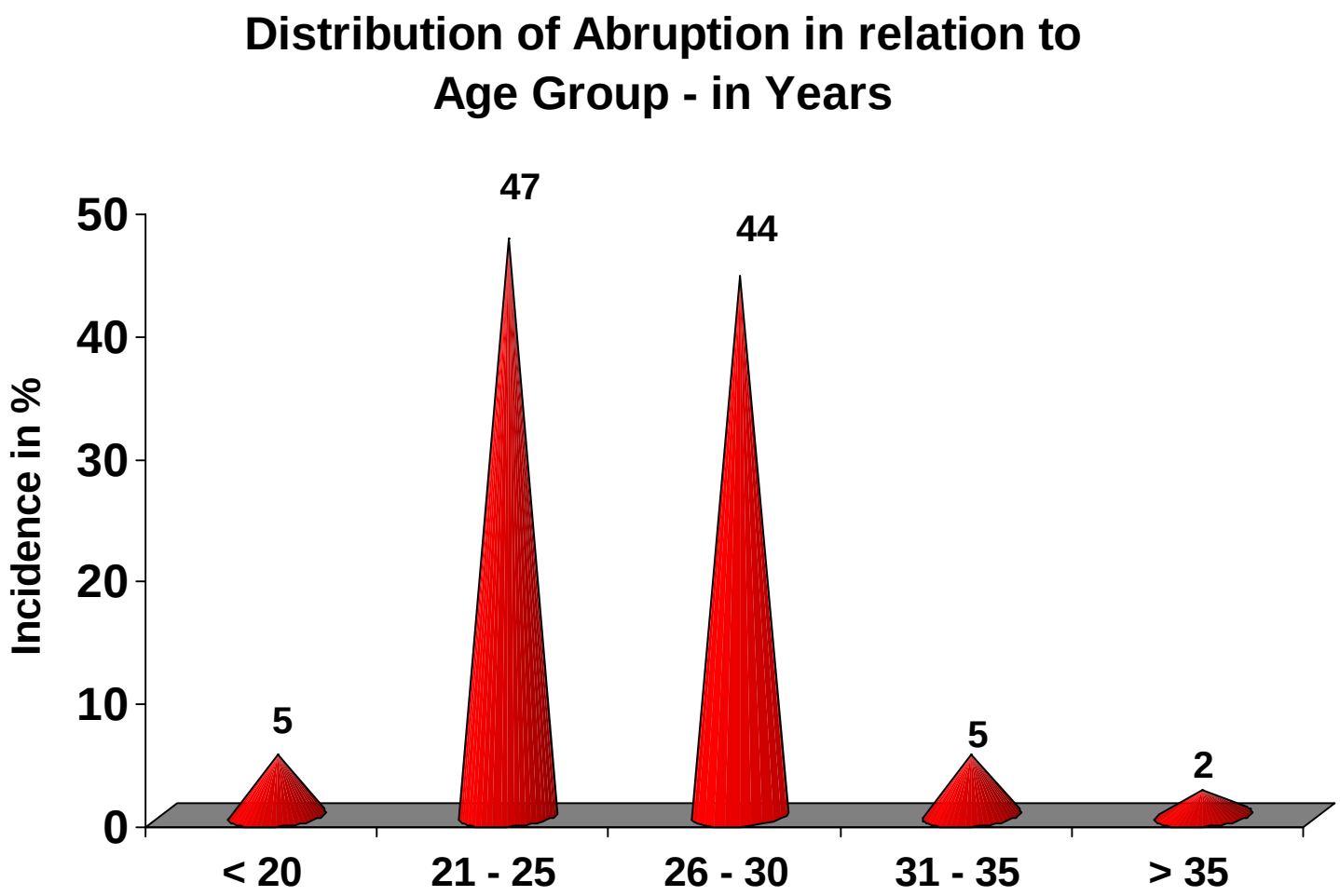
Distribution of Patients according to parity and the Incidence

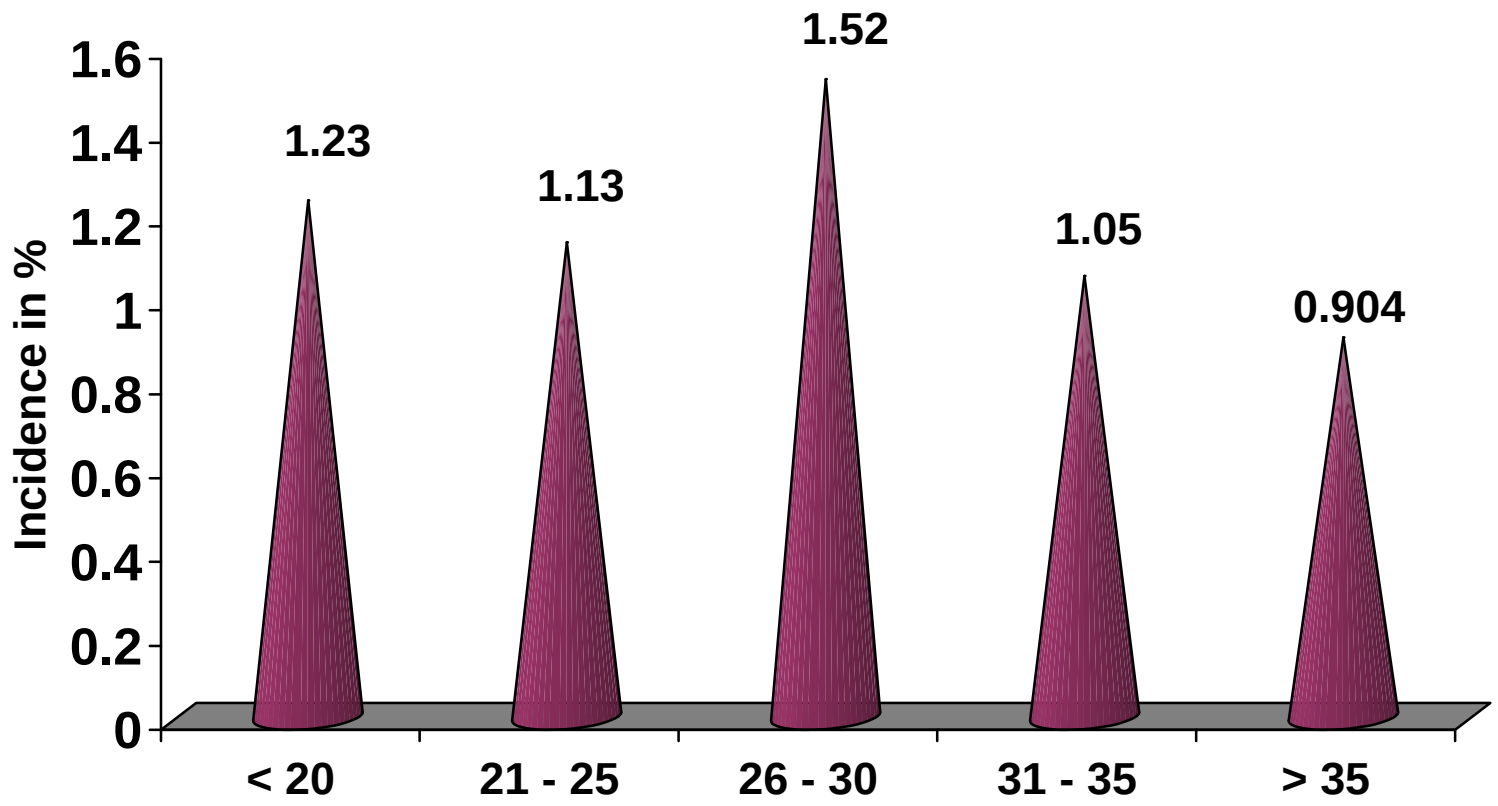
Parity	No. of Patients	Abruption	
		No.	Incidence
Primi	4,395	31	0.705 %
2nd Gravida	3,190	34	1.060 %
3rd Gravida	496	24	4.830 %
4th Gravida	37	14	37.830 %


Risk analysis of Parity
Incidence in %
45.00
40.00
35.00
30.00
25.00
20.00
15.00
10.00
5.00
0.00
1.00
2.66
3.87
43.93
1st Gravida
2nd Gravida
3rd Gravida
4th Gravida

Risk analysis of Parity Odds ratio

Parity	Odds ratio of abruption	Overall Risk
Primi	1.00	1.00
2nd Gravida	2.34	2.66
3rd Gravida	4.33	3.87
4th Gravida	35.84	43.93


Distribution of Abruption in relation to
Age Group - in Years
Incidence in %
50
40
30
20
10
0
47
44
5
5
2
< 20
21 - 25
26 - 30
31 - 35
> 35

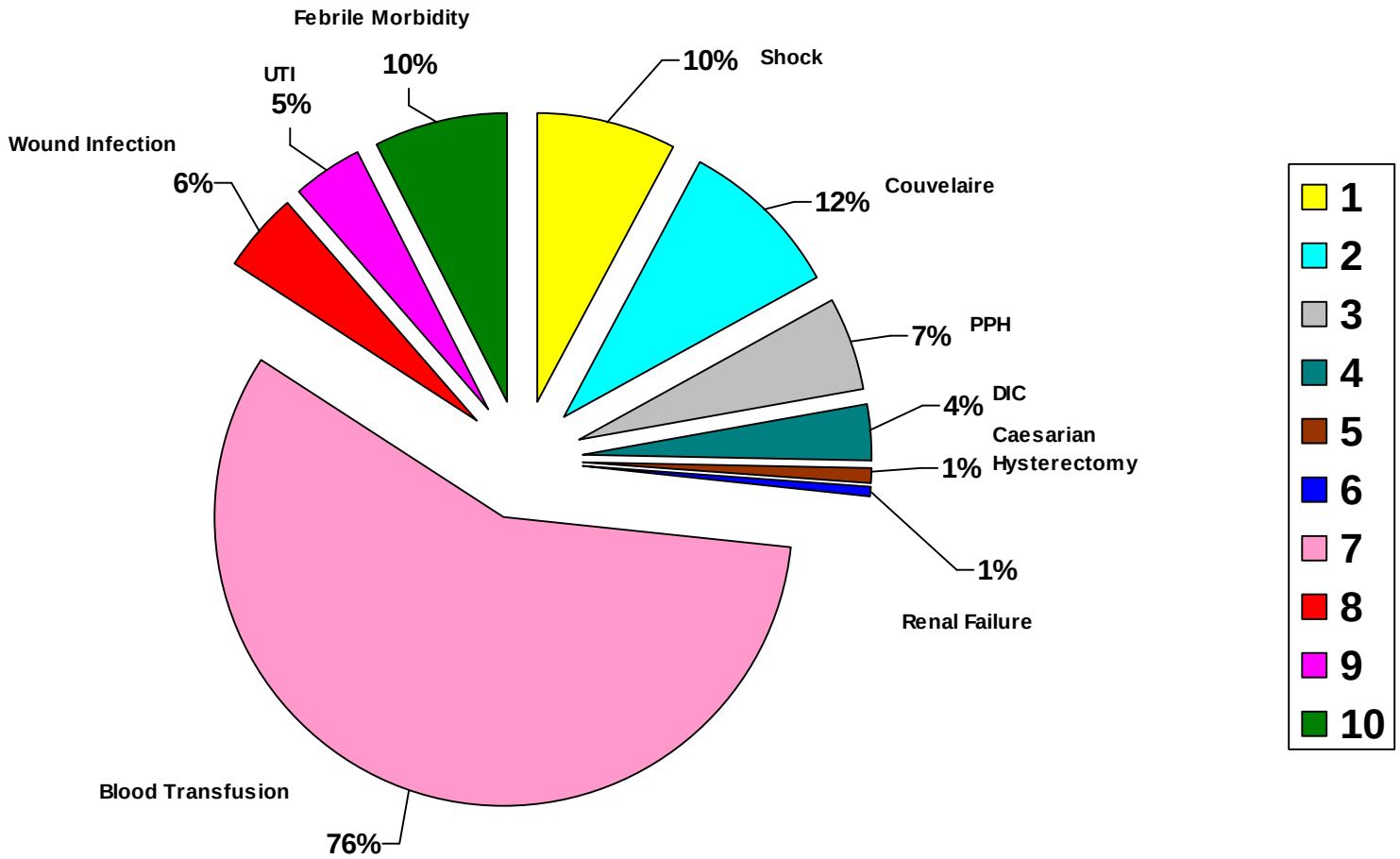

Relationship between Age group and Incidence of Abruption - in Years
Incidence in %
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
1.23
1.13
1.52
1.05
0.904
< 20
21 - 25
26 - 30
31 - 35
> 35

Distribution of Abruption in relation to Age Group

Age Group	Total no. of patients	Abruption	
		No.	Incidence
< 20 yrs.	404	5	1.23 %
21 – 25 yrs.	4,139	47	1.13 %
26 – 30 yrs.	2,882	44	1.52 %
31 – 35 yrs.	472	5	1.05 %
> 35 yrs.	221	2	0.904 %

Incidence of abruption is not significantly increasing with advanced maternal age in our studies.

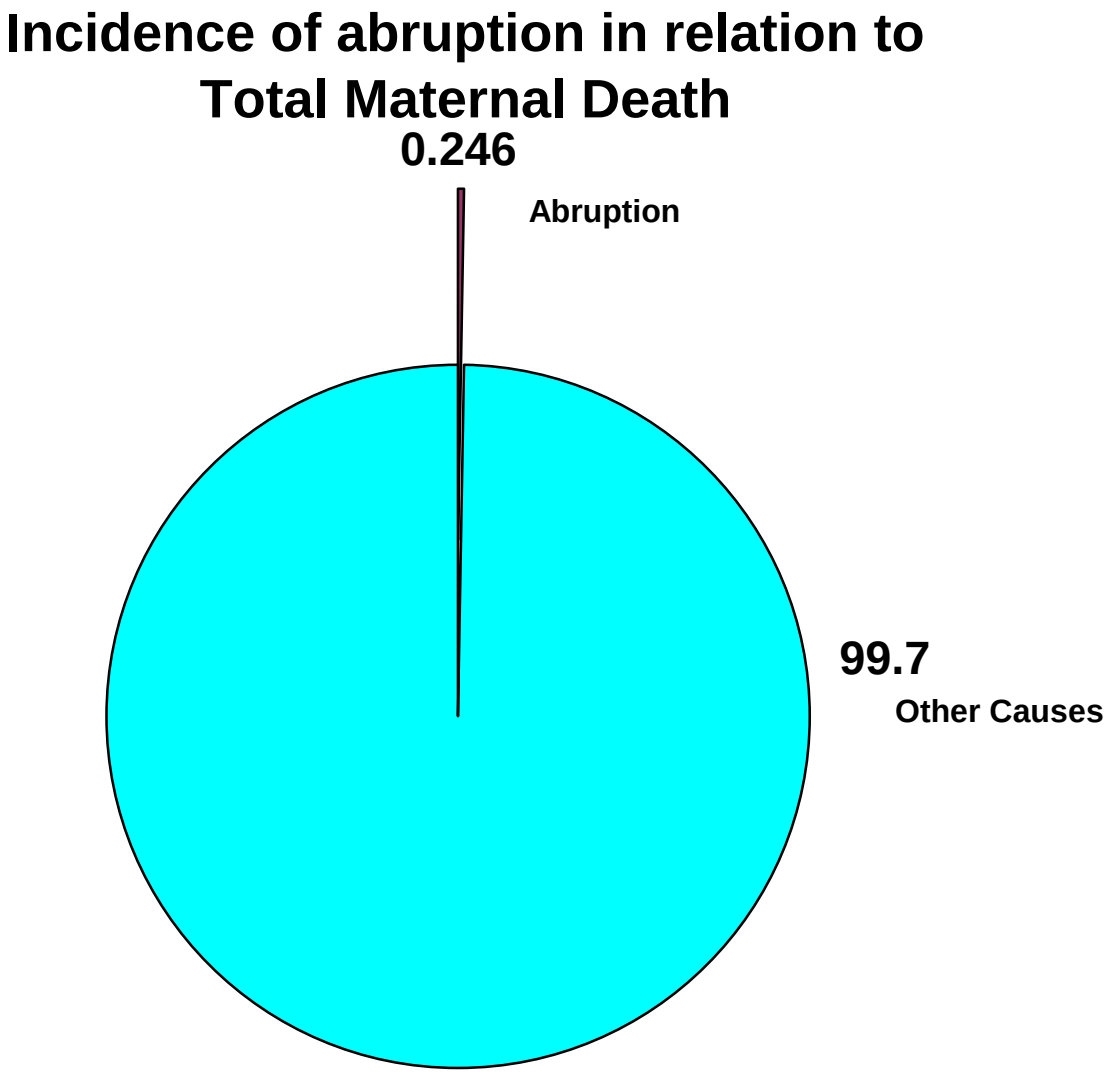
Maternal Morbidity associated with Abruption



Maternal morbidity associated with Abruption

Morbidity Status	No. of Cases	Incidence
Shock	9	10.4 %
Couvelaire uterus	10	12.1 %
PPH	6	7.2 %
DIC	3	3.61 %
Caesarian Hysterectomy	1	1.2 %
Renal Failure	1	1.2 %
Blood Transfusion	63	75.9 %
Wound infection	5	6.02 %
UTI	4	4.8 %
Febrile Morbidity	9	10.08 %

Blood transfusion is required in significant number of cases.

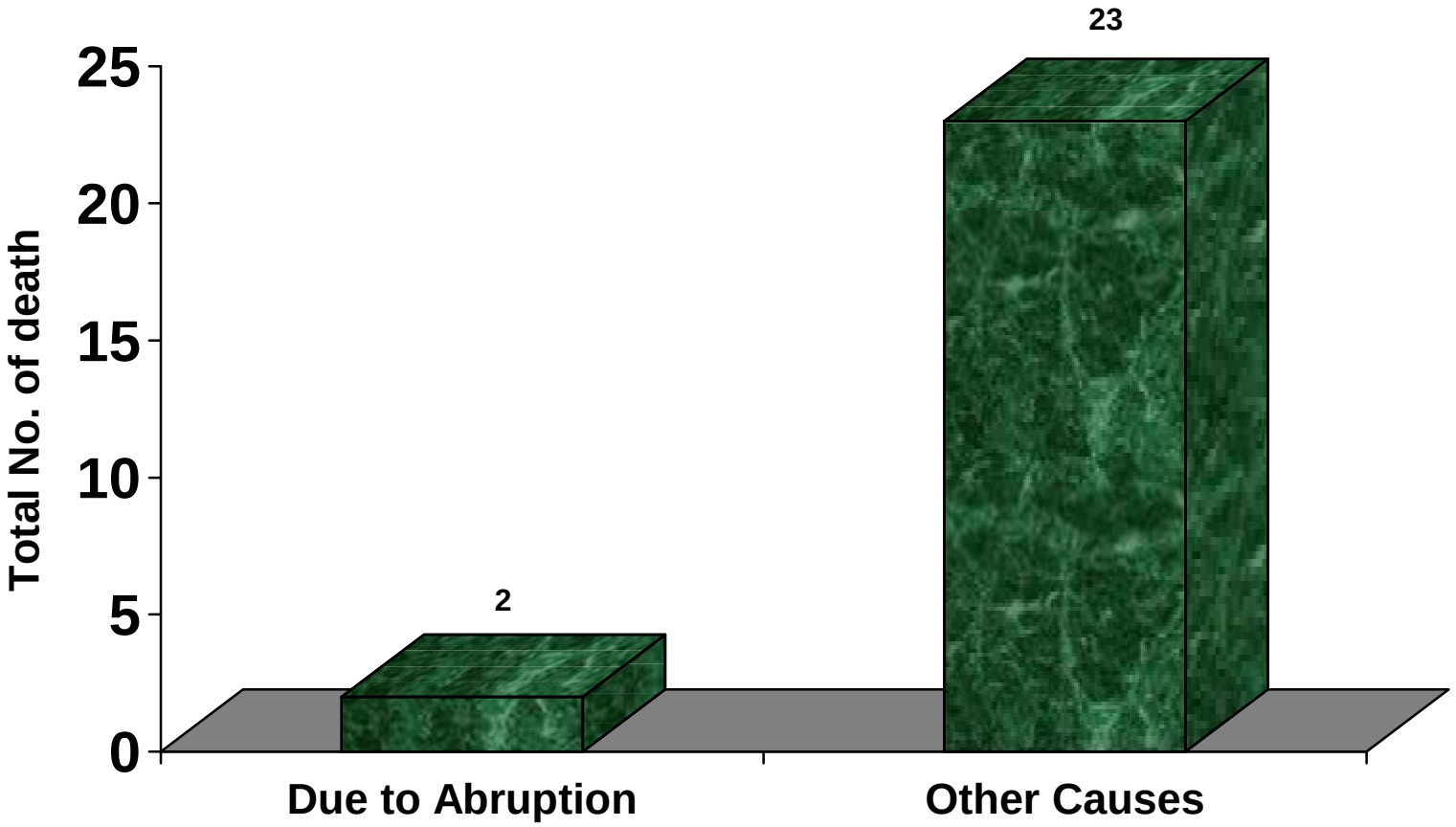

Incidence of abruption in relation to
Total Maternal Death
0.246
Abruption
99.7
Other Causes

Maternal mortality in abruption

Total No. of deliveries	No. of death due to abruption	Incidence
8,118	2	0.246 %

Incidence of maternal mortality in our hospital was 0.246 %

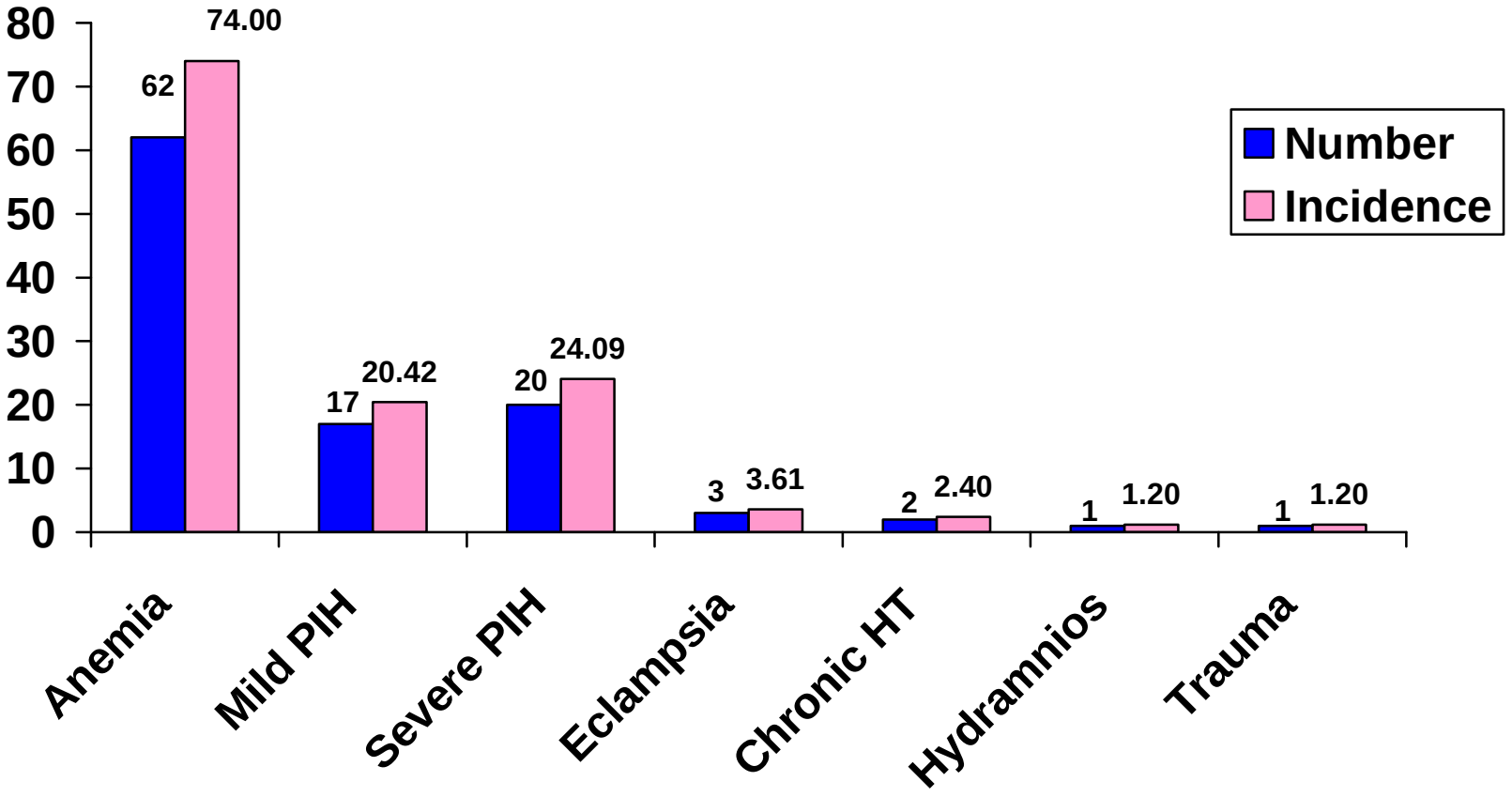
Contribution of abruption to total maternal death



Contribution of Abruption to total death

Total Death	Death due to Abruption	Incidence
25	2	8 %

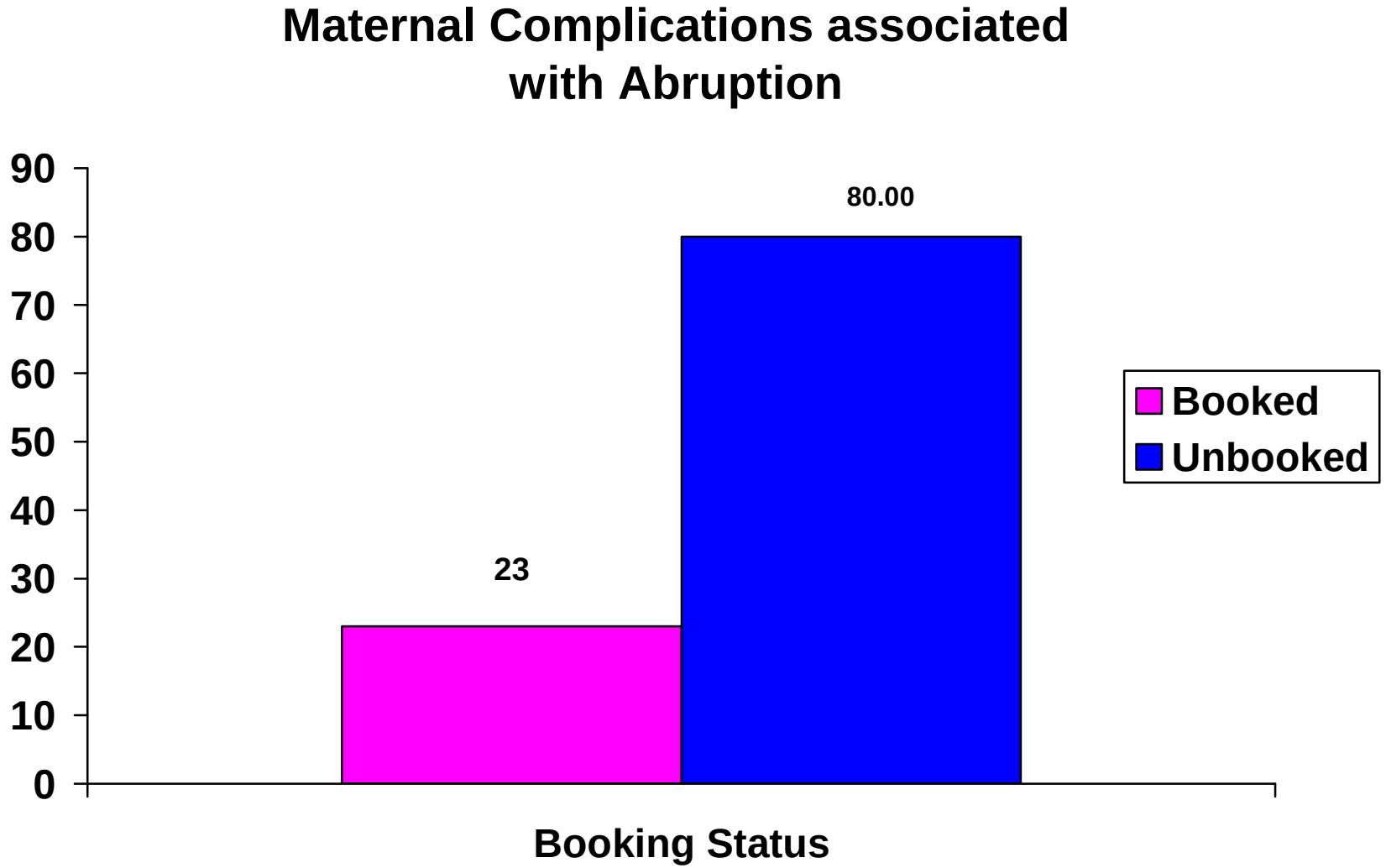
Maternal Complications associated with Abruption



Maternal complications associated with abruption

Complications	No. of Cases	Incidence
Anemia	62	74.00 %
Mild PIH	17	20.42 %
Severe PIH	20	24.09 %
Eclampsia	3	3.61 %
Chronic hypertension	2	2.4 %
Hydramnios	1	1.2 %
Trauma	1	1.2 %
Diabetes Mellitus	Nil	Nil
Heart disease	Nil	Nil

Hypertension disorders were associated with abruption in more than 50% of cases


Maternal Complications associated with Abruption
90
80
70
60
50
40
30
20
10
0
80.00
23
Booked
Unbooked
Booking Status

Maternal outcome in relation to socio-economic status

All the patients belong to low socio economic status.

Low - 103

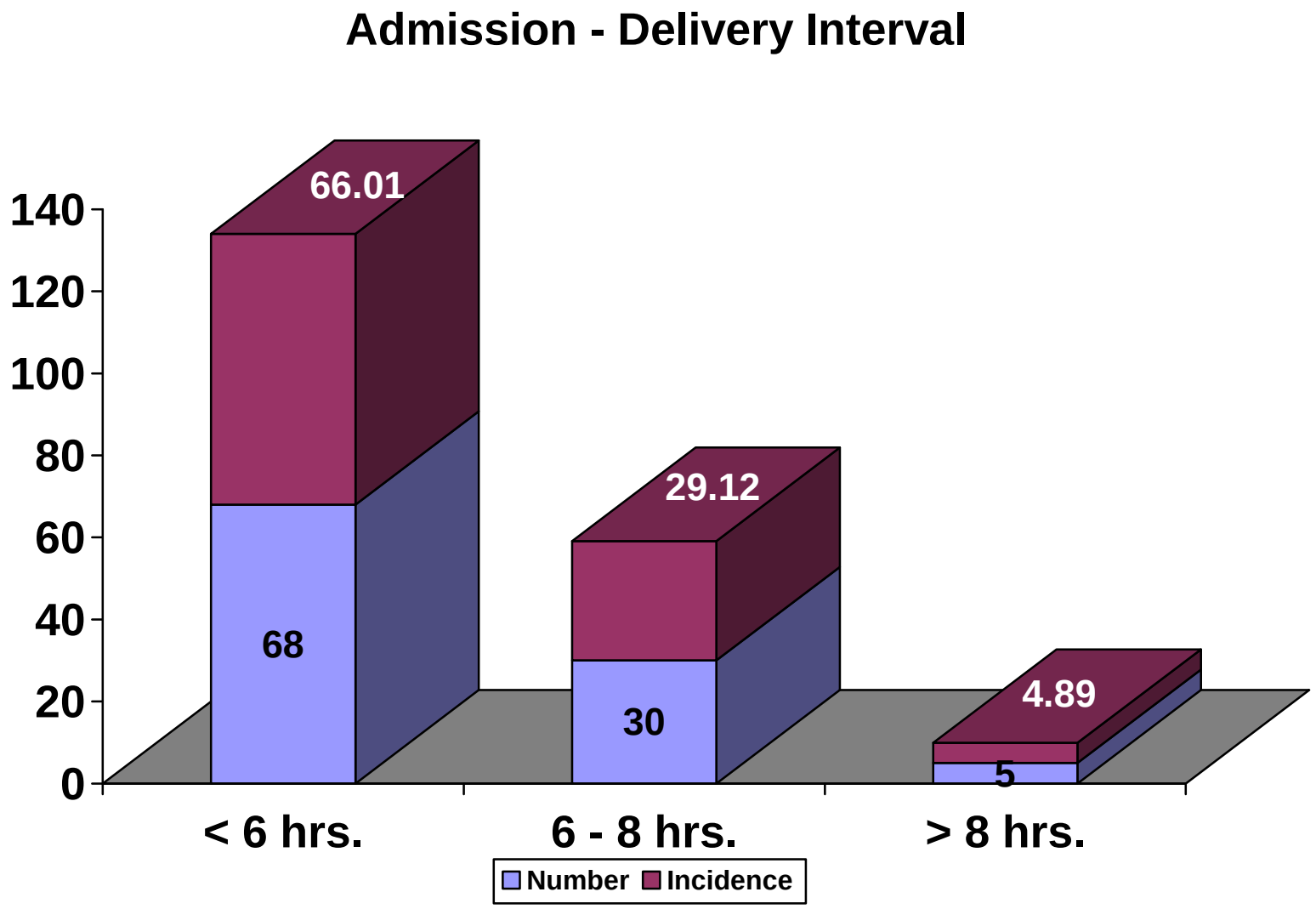
Middle - Nil

High - Nil

Booking status

Status	No. of Cases	Incidence
Booked	23	22.33 %
Unbooked	80	77.66 %

Maternal mortality and morbidity increased in Unbooked cased than the booked.

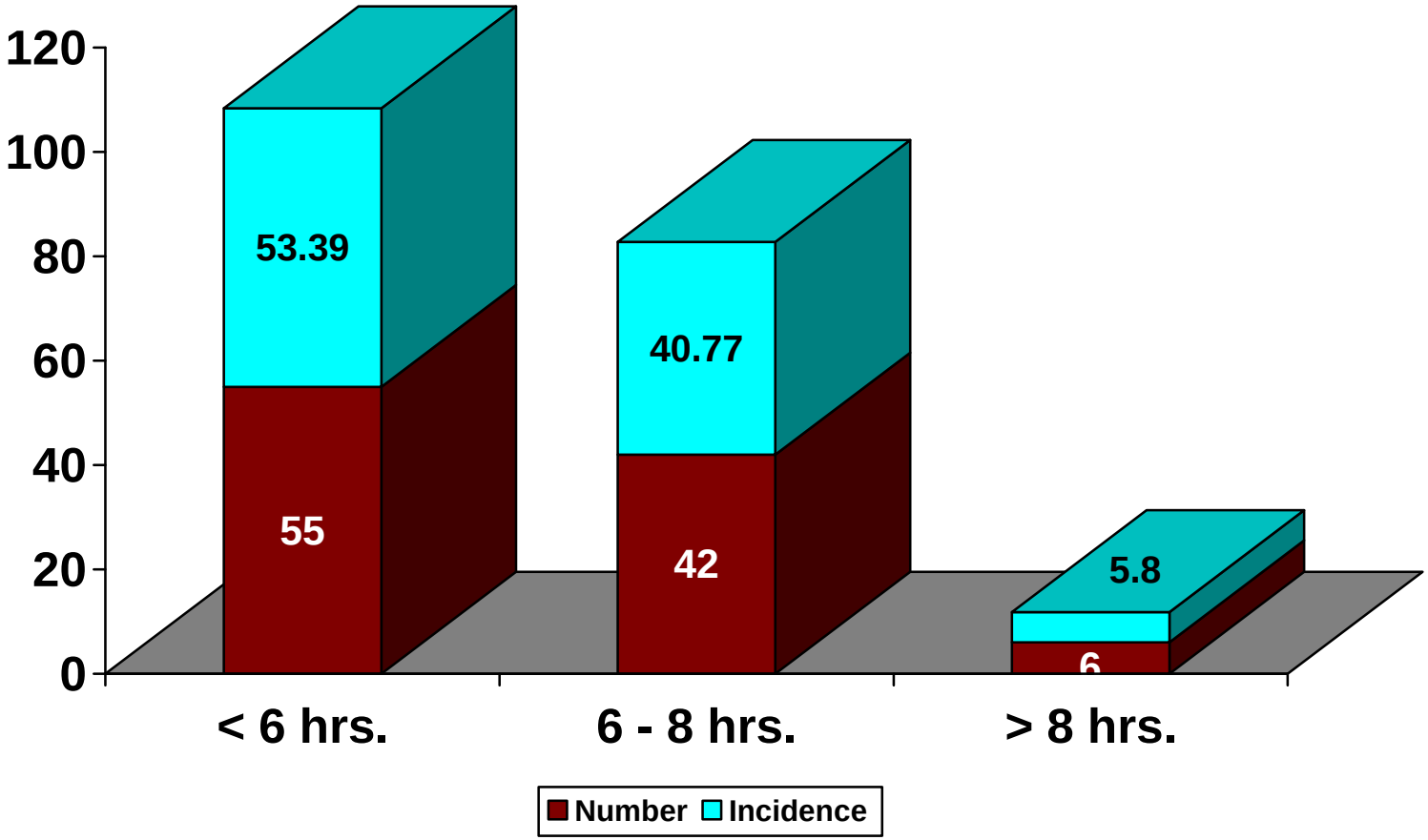

Admission - Delivery Interval
140
120
100
80
60
40
20
0
66.01
68
29.12
30
4.89
5
< 6 hrs.
6 - 8 hrs.
> 8 hrs.
Number
Incidence

Admission delivery interval

Duration	No. of Cases	Incidence
< 6 hrs.	68	66.01 %
6 – 8 hrs.	30	29.12 %
> 8 hrs.	5	4.85 %

Most cases of abruption are delivered quickly.

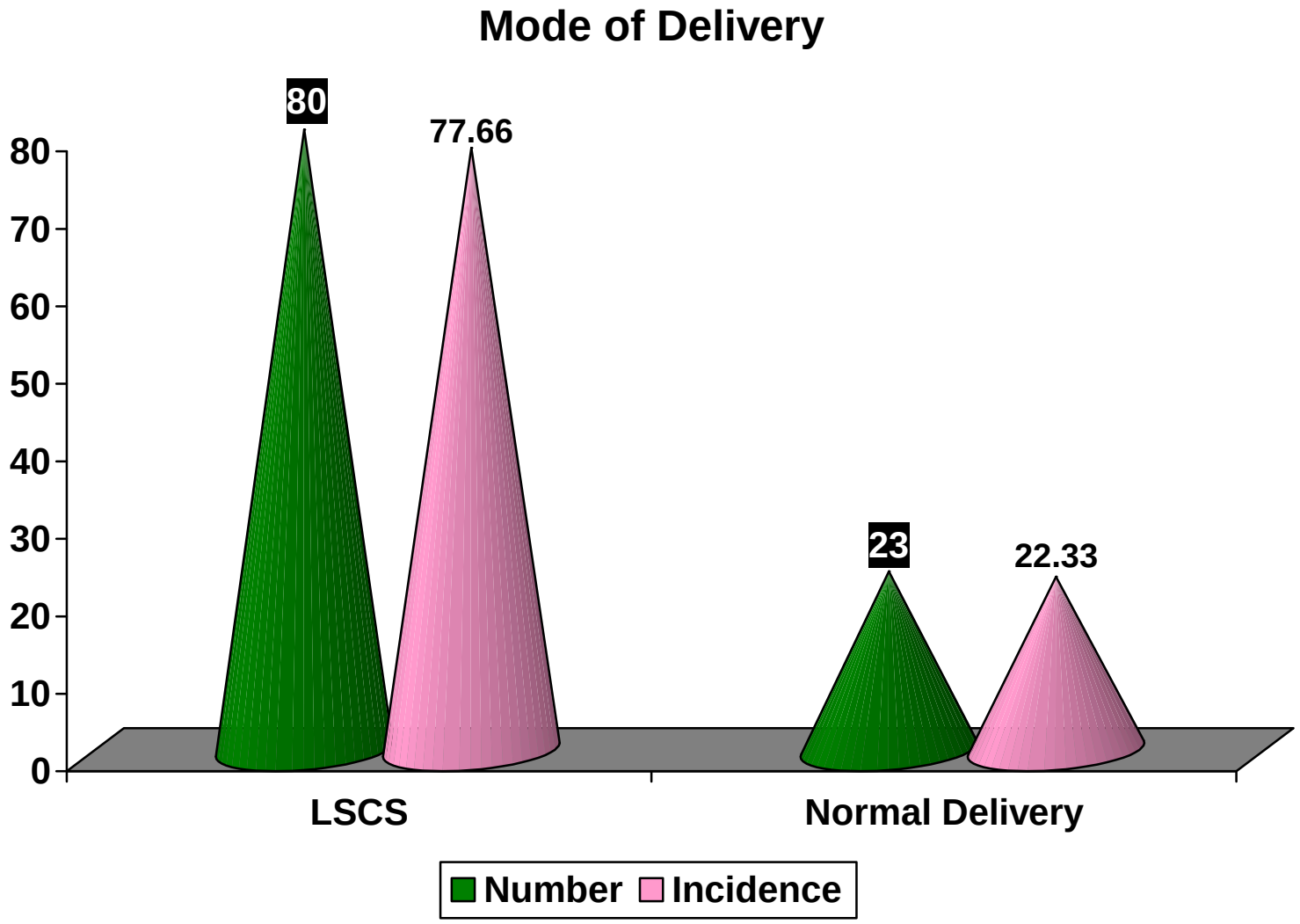
Abruption - Delivery Interval



Abruption delivery interval

Duration	No. of Cases	Incidence	Maternal Mortality Rate
< 6 hrs.	55	53.39%	-
6 – 8 hrs.	42	40.77%	-
> 8 hrs.	6	5.8%	2

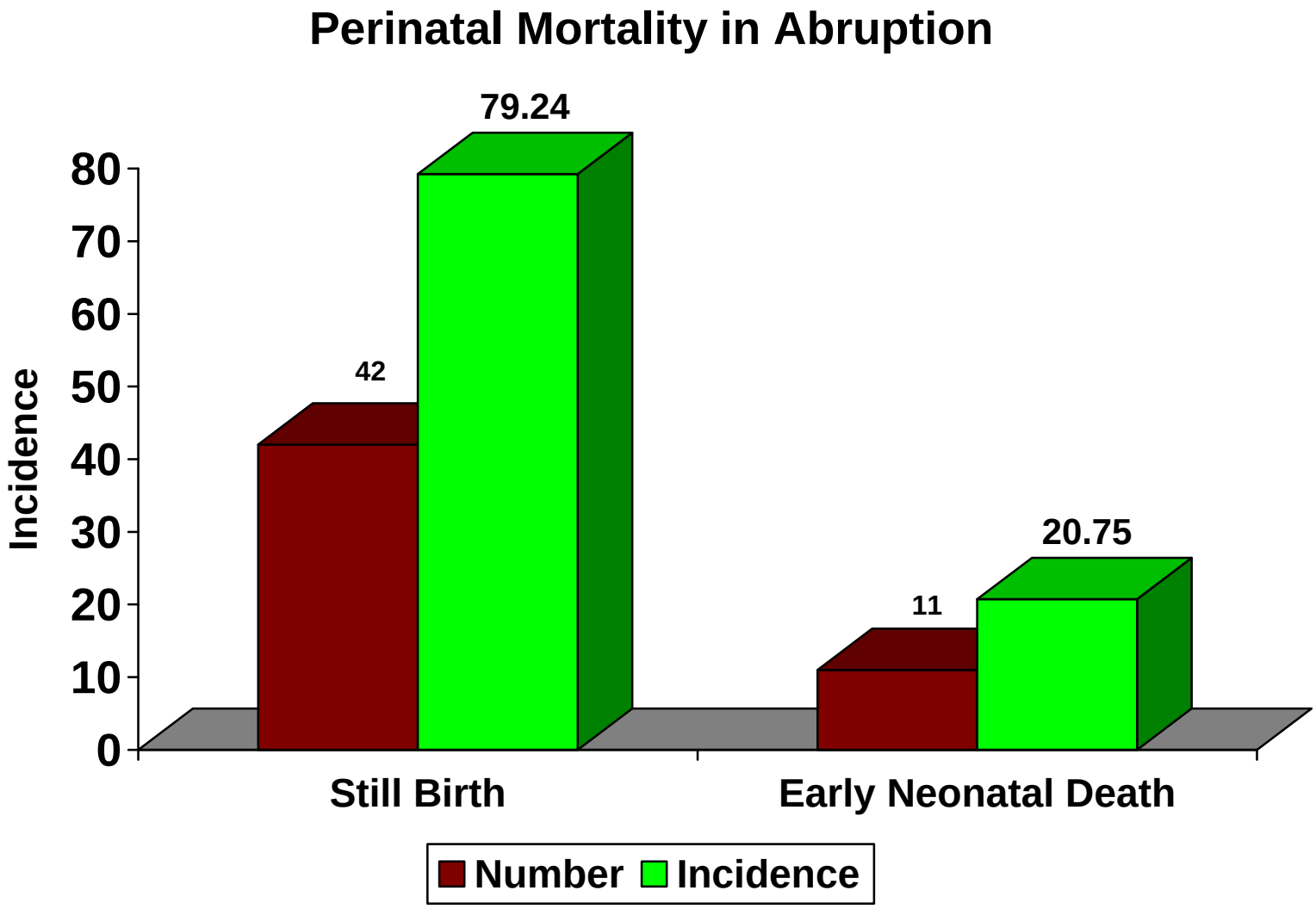
As abruption delivery interval prolong the maternal mortality rate increased.


Mode of Delivery
80
77.66
23
22.33
80
70
60
50
40
30
20
10
0
LSCS
Normal Delivery
Number
Incidence

Mode of delivery

Mode of delivery	No. of Cases	Incidence
LSCS	80	77.66%
Normal Vaginal delivery	23	22.33%

In our study we used liberal caesarian section. In accidental heamorrhage caesarean rates are almost 77%, this is done in order to reduce maternal complication and to improve fetal salvage (Bhatt 1989 and Walvekar 1998)


Perinatal Mortality in Abruption
79.24
42
20.75
11
80
70
60
50
40
30
20
10
0
Incidence
Still Birth
Early Neonatal Death
Number
Incidence

Perinatal Mortality in Annal Gandhi Memorial Govt Hospital, Trichy

Total Death - 487 cases

Death due to abruption - 53 cases

Incidence of death due to abruption - 10.88 %

Incidence of perinatal death in abruption

Total abruption - 103 cases

Perinatal death - 53 cases

Cause	Number	Incidence
Still Birth	42	79.24 %
Early neonatal death	11	20.75 %

The total perinatal deaths were 53 in our study of which 42 were still birth and 11 were early neonatal death.

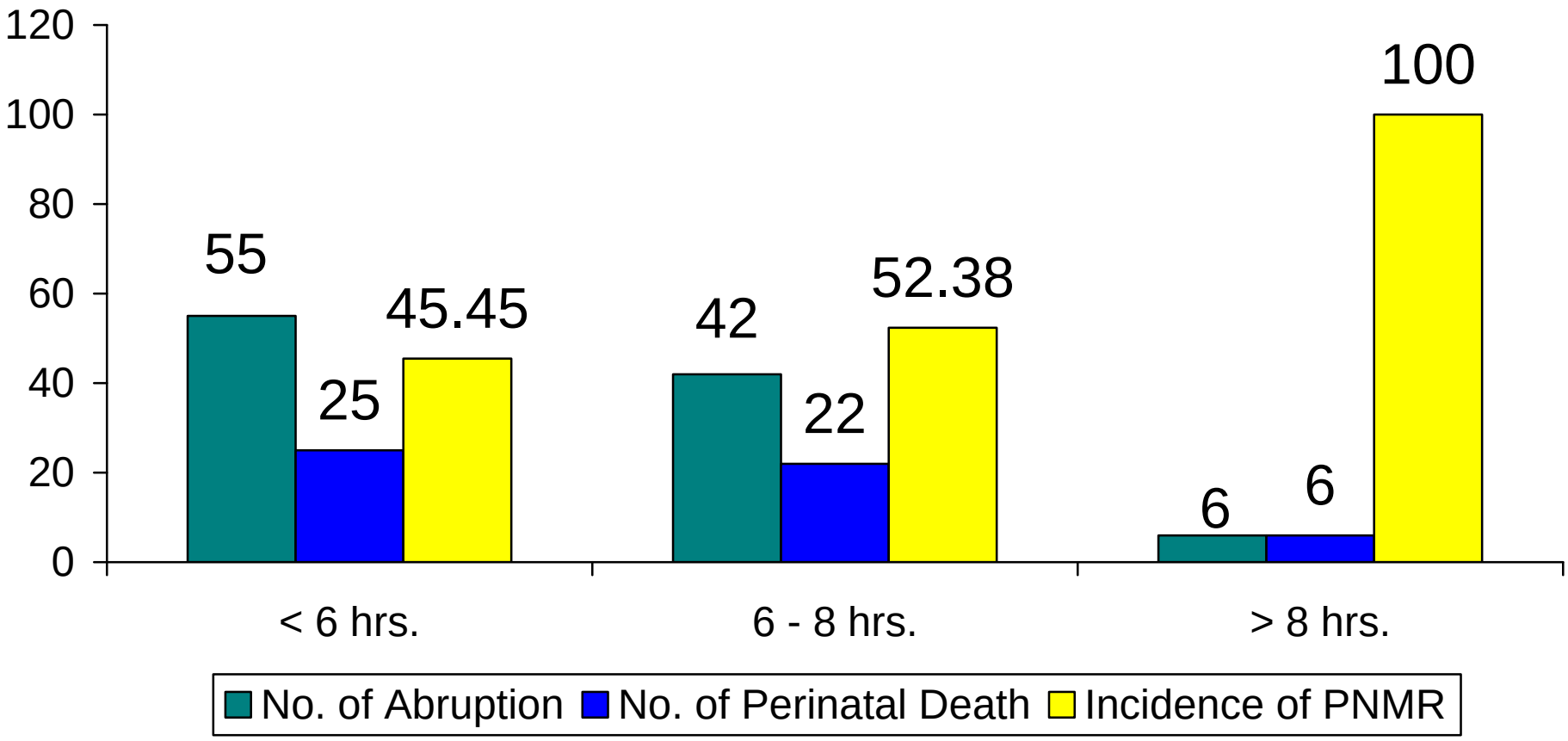
Perinatal mortality rate - 51.40 %

Contribution of perinatal mortality due to abruption in relation to total perinatal mortality

Total perinatal mortality - 487 cases

Abruption perinatal contribution - 53 cases

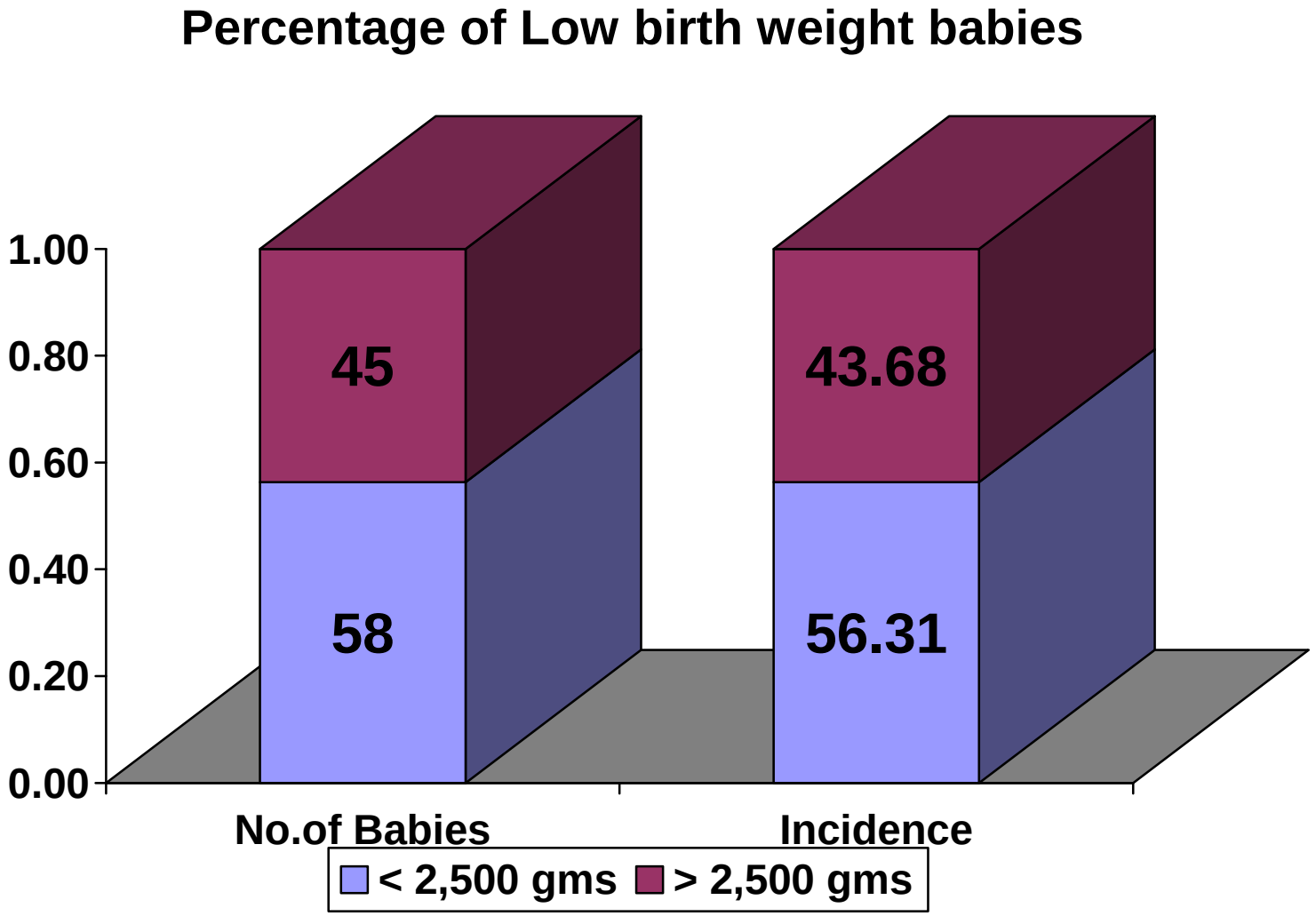
Abruption Delivery Interval in relation to PNMR



Abruption delivery interval in relation to perinatal mortality

Duration	No. of Abruption	No. of Perinatal Death	Incidence
< 6 hrs.	55	25	45.45 %
6 – 8 hrs.	42	22	52.38 %
> 8 hrs.	6	6	100.00 %

When the abruption delivery interval increase the perinatal mortality also increase.

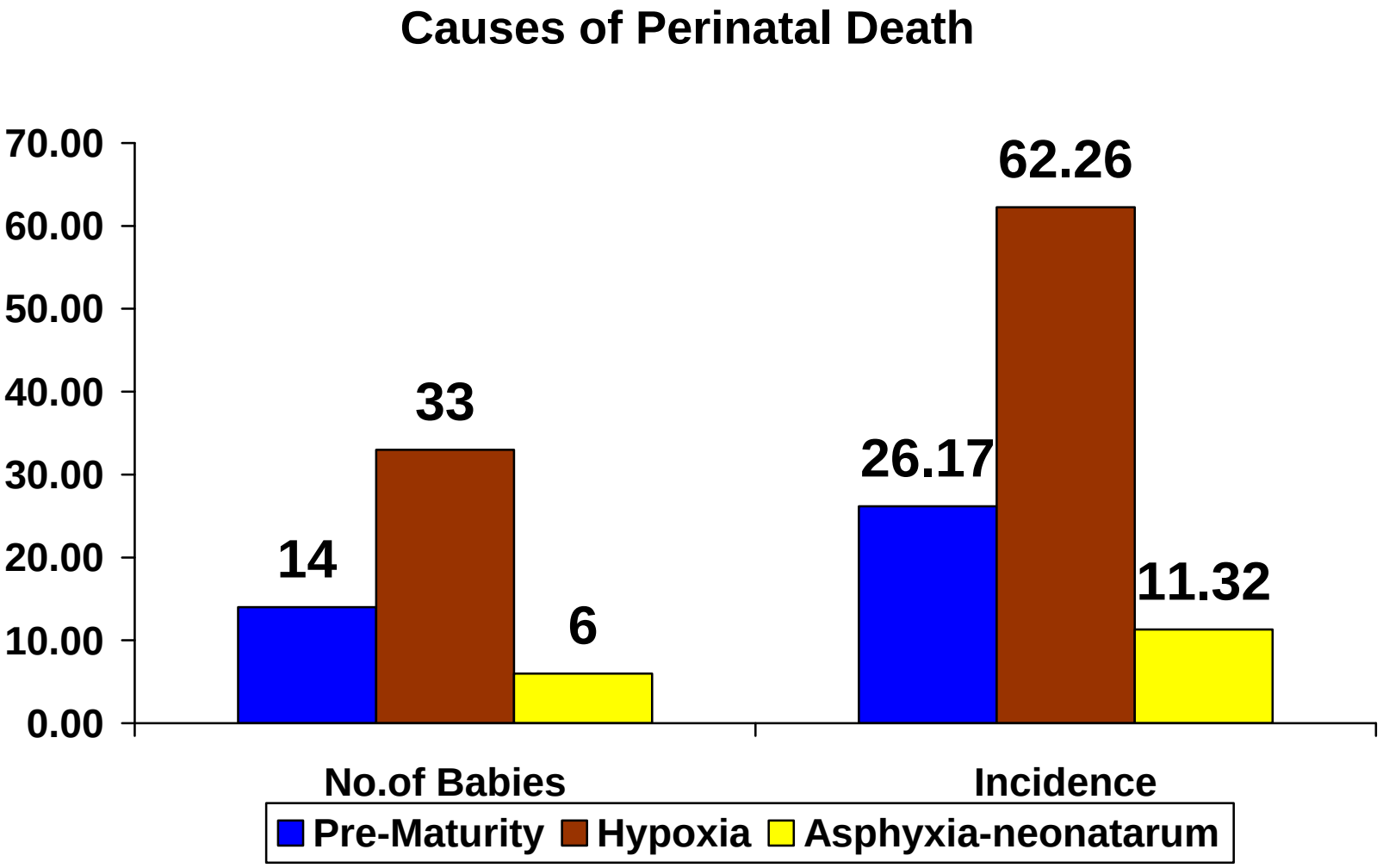

Percentage of Low birth weight babies
1.00
0.80
0.60
0.40
0.20
0.00
45
58
43.68
56.31
No.of Babies
Incidence
< 2,500 gms
> 2,500 gms

Birth weight and perinatal mortality – Relationship

Birth Weight	No. of Babies	No. of Perinatal Death	Incidence
< 1,500 gms.	6	6	100.00 %
1,500 – 2,000 gms.	23	14	60.80 %
2,000 – 2,500 gms.	29	14	48.27 %
2,500 – 3,000 gms.	25	15	60.00 %
3,000 – 3,500 gms.	14	3	21.42 %
> 3,500 gms.	6	1	16.66 %

Percentage of Low birth weight babies

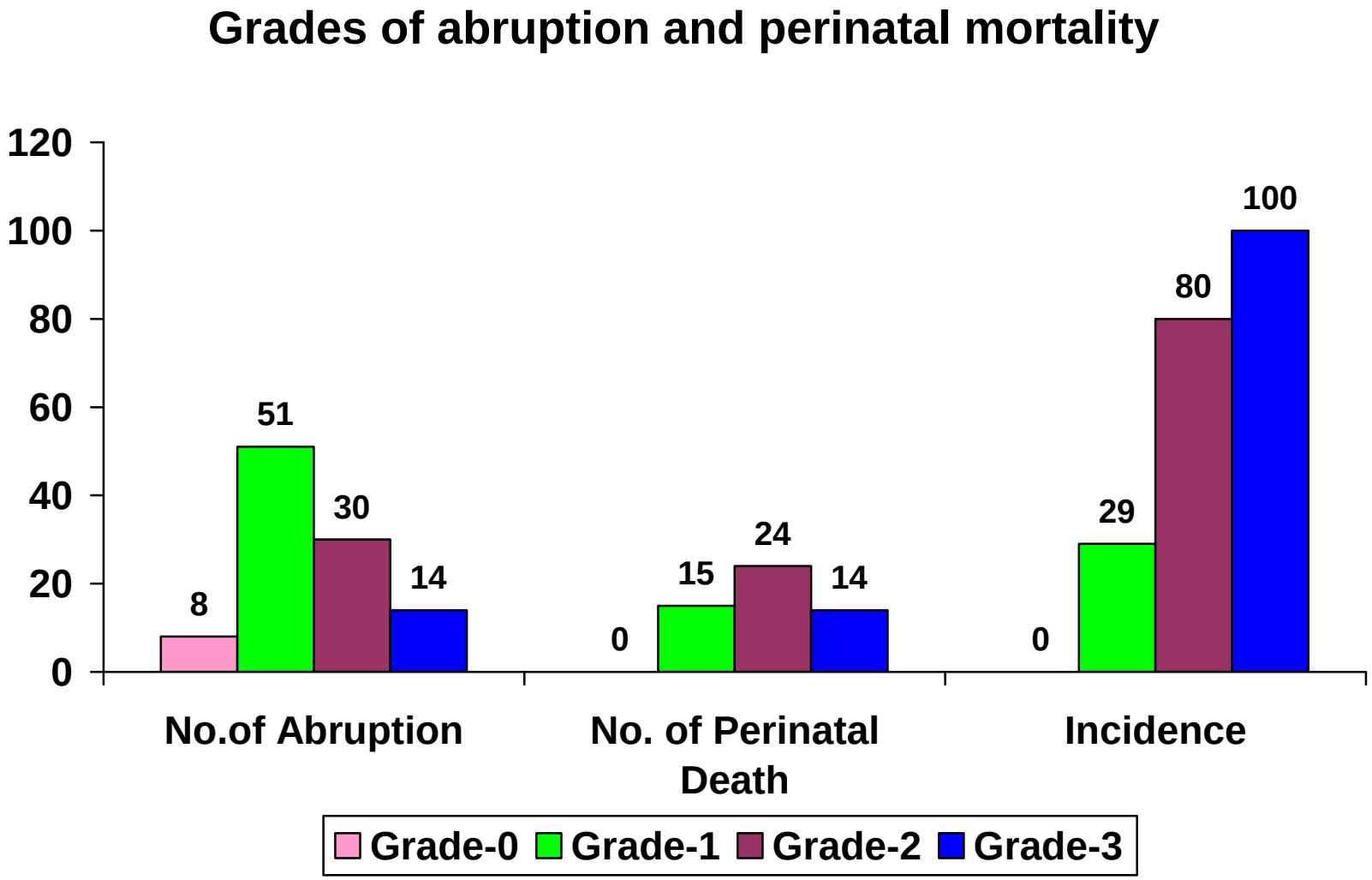
Birth Weight	No. of Babies	Incidence
< 2,500 gms.	58 cases	56.31 %
> 2,500 gms.	45 cases	43.68 %


Causes of Perinatal Death
70.00
60.00
50.00
40.00
30.00
20.00
10.00
0.00
14
33
6
26.17
62.26
11.32
No.of Babies
Incidence
Pre-Maturity
Hypoxia
Asphyxia-neonatarum

Causes of perinatal death

Causes of Death	No. of Babies	Incidence
Prematurity	14 cases	26.17 %
Hypoxia	33 cases	62.26 %
Asphyxia Neonatorum	6 cases	11.32 %

Hypoxia forms the major cause of death in abruption.


Grades of abruption and perinatal mortality
120
100
80
60
40
20
0
8
51
30
14
0
15
24
14
0
29
80
100
No.of Abruption
No. of Perinatal Death
Incidence
Grade-0
Grade-1
Grade-2
Grade-3

Grades of abruption and perinatal mortality

Grades	No. of Abruption	Percentage	Perinatal Death	Percentage
Grade 0	8	7.7 %	Nil	Nil
Grade 1	51	49.51 %	15	29.00 %
Grade 2	30	29.12 %	24	80.00 %
Grade 3	14	13.59 %	14	100.00 %

Perinatal mortality is proportional to severity of abruption.

DISCUSSION

The incidence of abruption in the present study is 1.26 %. i.e. 1 in 79 cases. It is slightly higher than reported by Amarnath (0.04%) in 1990, but significantly lower than reported by Hibbard 3.79 %.

In our study 95% of CI was 0.88 – 1.19 meaning that if the study is conducted by some other person with the same sample size we can expect an incidence of 1%. As per the previous tables the incidence of abruption in the previous year is also around 1.2%.

Incidence of abruption in various studies

Menon	1963	1.80 %
Hibbard	1969	3.76 %
Cotten	1986	1.22 %
Krohn	1987	0.65 %
Sholl	1987	0.80 %
Amarnath	1990	0.40 %
GMH Egmore	1997	0.70 %
Iyasu	1993	0.60 %

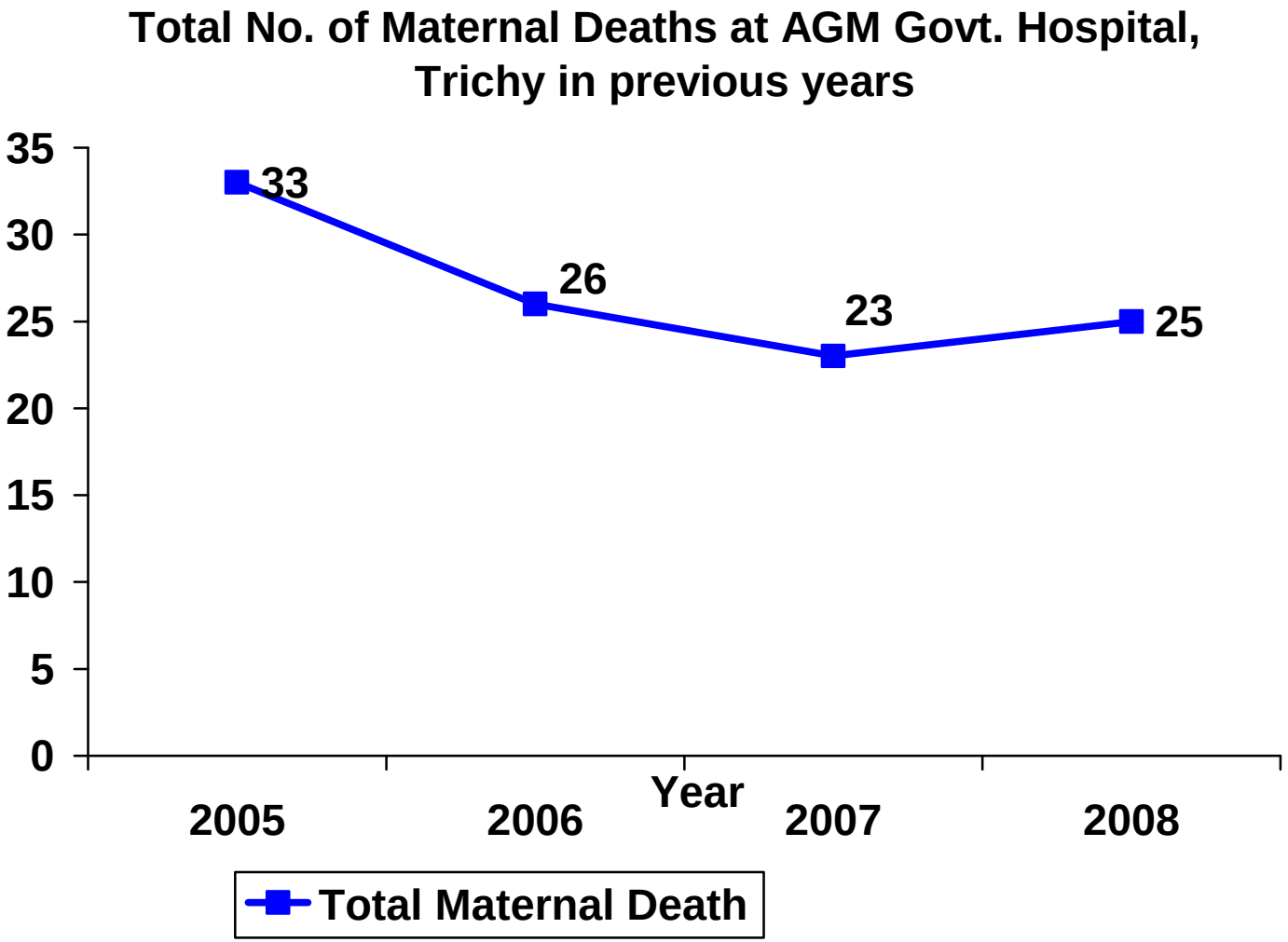
The incidence of abruption in our studies is higher than that reported by Menon - 1963, Hibbar – 1969 and Sholl – 1987.

Regarding the relationship of parity with incidence of abruption from our study we found as the parity increases the incidence increases. P Value is significant. Para 4 has odds ratio of 36 in abruption. This correlates the studies of Pritchard and associates (1991) and Babinski and Collobration (1999).

In our studies advanced maternal age was not significantly associated with an increase in the incidence of abruption as observed by Frederickson and associates in 1999. We had two cases of maternal death among 103 cases. Incidence is 0.25%, mortality in this case was due to DIC / ARF.

Maternal Mortality

Out of 8,118 cases there were 25 maternal deaths. Since, the safe motherhood initiative began more than a decade ago. There have been a number of important developments in the international women's health field. There are atleast three ways in which maternal mortality is special. First, its magnitude, second its epidemiologic nature, and third, its programmatic requirements. There are more than half a million maternal deaths occur in every year, 99% of which are in developing countries


Total No. of Maternal Deaths at AGM Govt. Hospital, Trichy in previous years
35
30
25
20
15
10
5
0
33
26
23
25
2005
2006
2007
2008
Year
Total Maternal Death

with 16% of World's population, India accounts for 20% of the World's maternal death. The rate is 5.4 / 1,000 which are very high compared to other countries in Asia.

MMR in abruption according to various authors

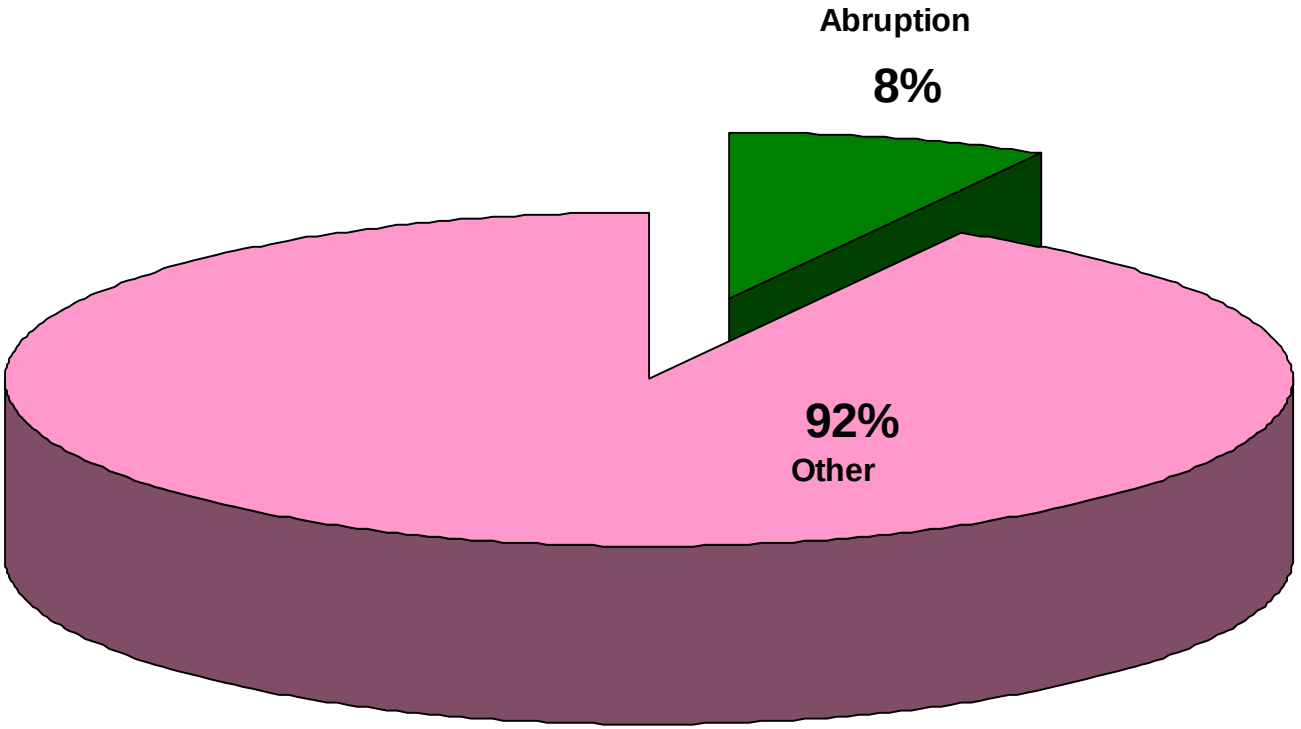
Author	Year	Incidence
Prema Naidu	1960	1.80 %
Podder	1961	6.40 %
Menon and Mudaliar	1972	28.00 %
Krauss T	1993	0.80 %
AGM Govt.Hospital. Trichy.	2008	0.012 %

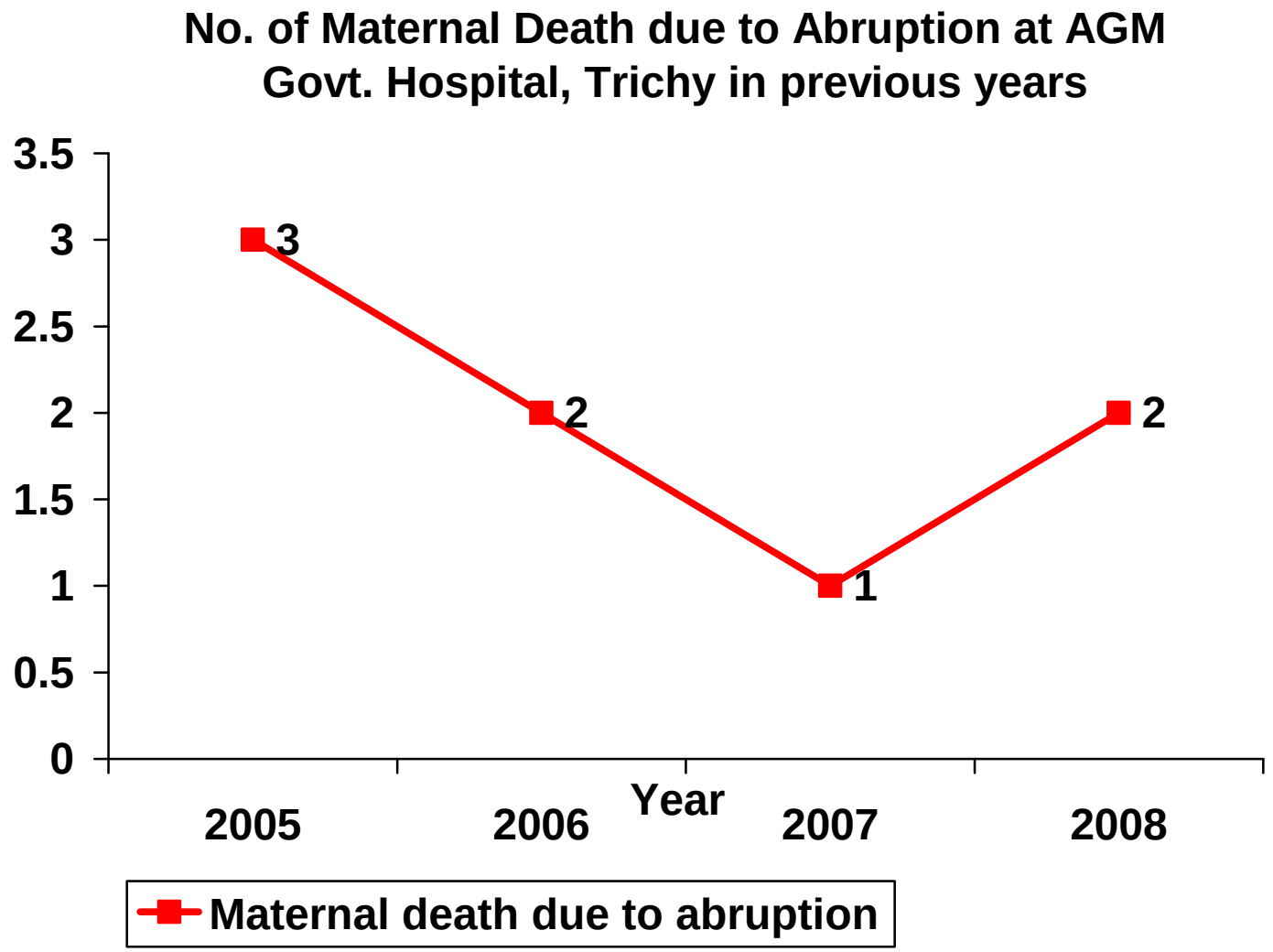
The total maternal deaths in our hospital were 25, deaths due to abruption were 2, and contribution to maternal deaths was 8%.

Incidences of Maternal mortality rates in previous 3 yrs.

Year	No. of Deliveries	No. of Maternal Death
2005	8,990	33
2006	8,250	26
2007	7,927	23

Incidence of abruption
Total Death




No. of Maternal Death due to Abruption at AGM Govt. Hospital, Trichy in previous years
3.5
3
2.5
2
1.5
1
0.5
0
3
2
1
2
2005
2006
2007
2008
Year
Maternal death due to abruption

Causes of Maternal Mortality in Previous years

Year	Causes of Death	No. of Cases
2005	DIC	3 cases
2006	Renal Failure	2 cases
2007	PPH	1 case
2008	DIC / RF	2 case

Causes of maternal mortality in the previous years are mainly due to disseminated intravascular coagulation (DIC), postpartum hemorrhage and acute renal failure. Regarding the maternal morbidity in abruption cases, shock was presenting in more number of cases. Renal failure present in one case (1.2%), since the patient had associated DIC, we couldn't able to revive the patient. The current incidence of renal failure is difficult to asses due to lack of information in definition. DIC was seen in 3 cases, the incidence are severe enough to kill the fetus according to William's 21st edition are 30% in abruption.

In our study the incidences of DIC are 3 cases (3.61 %), according to Menon the incidence of coagulation failure in abruption was 24.6%. Couvelaire uterus was present in 12% of our cases. Correct incidence of Couvelaire was not known because laparotomy was not done in all cases.

Urinary tract infections are slightly higher due to prolonged catheterization. When bleeding in abruption is concealed maternal morbidity was increased.

Hypertension was present in almost 50% of our cases. Purandare observed 47% of abruption cases were associated with hypertension, in our study it was higher. The percentage of unbooked cases in our hospital series was 77.2%. Perinatal mortality and morbidity was significantly increased in unbooked cases. The maternal mortality in our case was also an unbooked case. Maximum number of patients came from low socio economic status as also reported by Valere et al (1968) and Brenner (1978).

When abruption admission interval and abruption delivery interval is increased the perinatal mortality was significantly increased. From our studies of admission delivery interval, it is observed most of the abruption was delivered within a period of 6 hrs. Hence the perinatal mortality was not dependent as admission delivery interval in our studies.

There are two maternal deaths in abruption in our hospital is due to DIC / ARF which was referred cases delivered more than 8 hrs. Hence the admission delivery interval did not affect the maternal mortality in our series. Incidence of low birth rate in our series was 56.31%. Regarding the grade of abruption and perinatal mortality, the perinatal

mortality was directly proportional to the grade of abruption. Maternal morbidity is also increased with increase in the severity of abruption.

Ultrasonogram placental localization was accurately made in 78% of cases. Caesarian section rate was higher with an incidence of 84.33%. As for as maternal morbidity is concerned the number of patients who require blood transfusion are increased in abruption. Das (1975) reported blood transfusion in 35% of cases which is much lower than observed in our series of about 75.9%.

Perinatal Morbidity and mortality

In our study we had total death of about 487 babies of which 53 babies died due to abruption and the main cause being hypoxia. The perinatal morbidity and mortality is less in minor degrees of abruption. However successful labor and vaginal delivery confer advantages over babies delivered by caesarean section in term of respiratory function. The risk of perinatal morbidity and mortality is dependent on the gestation, with a significant reduction in the neonatal respiratory morbidity for each advancing week of gestation from 37 to 40 weeks babies (Morrison et al 1995 BJOG 2002).

SUMMARY

1. During one year study period there was 8,118 patients, of which we had 103 cases of abruption.
2. In our study almost 77.66% were unbooked, maternal and perinatal morbidity and mortality was significantly increased.
3. Incidence of abruption among total deliveries was 1.26%.
4. There is definite increased incidence of abruption in females of greater parity.
5. There is no significant increased incidence with advanced maternal age.
6. Maternal mortality was 0.25% in our studies.
7. Contribution of abruption deaths in total maternal deaths was 8%.
8. Perinatal mortality and morbidity was increased as the abruption delivery interval was increased.
9. Major cause of perinatal mortality in abruption was due to hypoxia.
10. Incidence of caesarian section in abruption was 77.66%.
11. Hypertension was present in more than 50% cases of abruption.
12. Blood transfusion was required in more than 75.9% of cases.

CONCLUSION

Abruption is still one of the most serious obstetric emergencies. Aetiology remains obscure in many cases and it often present without warning. Fortunately maternal mortality from abruption was reduced considerably due to implementation of good obstetric care and blood transfusion services. But, it is still an important cause of maternal morbidity and mortality and perinatal loss. Elucidation of precise history, etiology and prevention especially abruption remains the principal challenge of future.

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PROFORMA

Name

Age

Husband Name

IP No.

Booked / Unbooked

Socio Economic Status

Marital history

Married since

Consanguinity

Menstrual History

LMP

EDD

Menstrual cycle

Obstetric History

Gravida

Para

No. of previous abortion

1. Spontaneous abortion
2. Induced abortion
3. History of D and C

No. of children alive

LCB

Previous mode of deliveries

LSCS

Vaginal delivery

Previous history of abruption

Contraception

Past surgical history

Myomectomy

Past Medical history

Hypertension

Diabetes

Heart disease

Previous history in present pregnancy

History of PIH

History of bleeding in 1^{st} and 2^{nd} trimester

History of hospitalization

History of previous blood transmission

Family history

History of diabetes

History of hypertension

Personal History

Smoker

Alcoholic

Symptoms

1. Amenorrhea

2. Pain – onset, duration

3. Bleeding – onset, duration

4. Rupture of membrane

5. Diminished fetal movements

6. Loss of fetal movements

General Examination

Sensorium

Anemia

Jaundice

Edema

Vital signs – Temperature,
pulse,
BP, RR

Systemic Examination

Cardio vascular system

Respiratory system

Obstetric Examination

Fundal height

Over distended uterus

Tense, tender

Acting

Presenting part

Fetal heart rate

External Genitalia

Normal or abnormal

Speculum examination

Local cause

Per vaginal examination

Bishop score

Color of the liquor

Diagnosis

Investigations

Hemoglobin

Bleeding time, clotting time

Blood grouping and RH typing

Clot retraction time

Serum fibrinogen

Liver function test

Renal function test

Blood sugar

Ultrasound

Gestational age

Retro placental clot

Placental localization

Liquor

Congenital anomalies

Treatment

Rest

Sedation

Steroid

Oxytocin augmentation

Artificial rupture of membrane

Antibiotic

IV fluids

Blood transfusion

Plasma transfusion

Maternal outcome

1. Gestational age at delivery
2. Mode of delivery

   LSCS

   Vaginal delivery

   Forceps

Findings at LSCS

Retro placental clots

Couvelaire uterus

Maternal complications

Shock

Renal failure

DIC

PPH

Death

Baby

Status-Live, Still born,Death

Weight

Apgar score

MASTER CHART

S No	Name	Age	Booked Status	Obstetric code	Diagnosis	Mode of Delivery	Baby weight	Apgar score	Still born	Neonatal death	Alive	Maternal mortality	Maternal morbidity	Maternal morbidity
1.	Easwari	39	UB	$G_4P_3L_3$	Abruption-Grade-1	VD	1.2	4 / 10 7 / 10	-	+	-	-	UTI	
2.	Muthukannu	20	UB	Primi	Abruption-Grade-2	LSCS	1.7	7 / 10 8 / 10	+			-	BL TR	
3.	Balammal	24	UB	Primi	Abruption-Grade-1	LSCS	3.1	8 / 10 9 / 10			+	-	BL TR	
4.	Mohana	25	UB	$G_2P_1L_1$	Abruption-Grade-3	LSCS	1.4	0	+			-	Shock	DIC
5.	Valli	24	Booked	$G_3P_2L_2$	Abruption-Grade-2	VD	3.6	8 / 10 9 / 10			+	-	BL TR	
6.	Annakili	27	UB	$G_2P_1L_1$	Abruption-Grade-1	LSCS	3.5	8 / 10 9 / 10	+			-	BL TR	
7.	Palaniammal	28	UB	$G_3P_2L_1$	Abruption-Grade-0	LSCS	1.9	6 / 10 8 / 10			+	-	WI	
8.	Malarkodi	23	UB	Primi	Abruption-Grade-3	LSCS	1.6	0	+			-	Shock	CU
9.	Muthammal	40	UB	$G_2P_1L_1$	Abruption-Grade-1	LSCS	3.7	8 / 10 9 / 10			+	-	BL TR	
10.	Jeyanthi	34	Booked	$G_3P_2L_2$	Abruption-Grade-2	LSCS	1.8	5 / 10 7 / 10			+	-	BL TR	
11.	Saraswathi	27	UB	$G_4P_3L_3$	Abruption-Grade-1	VD	2	4 / 10 7 / 10		+		-	BL TR	

12.	Kumutha	29	UB	$G_2P_1L_1$	Abruption-Grade-2	LSCS	1.3	0	+			-	Shock	
13.	Parameswari	25	Booked	Primi	Abruption-Grade-1	LSCS	2	4 / 10 7 / 10			+	-	WI	
14.	Johsi Parimala	21	UB	Primi	Abruption-Grade-2	LSCS	1.9	5 / 10 6 / 10	+			-	BL TR	
15.	Sumathi	23	Booked	Primi	Abruption-Grade-1	LSCS	2	4 / 10 7 / 10			+	-	BL TR	
16.	Lakshmi	27	UB	$G_2P_1L_1$	Abruption-Grade-2	LSCS	2	4 / 10 7 / 10			+	-	BL TR	CU
17.	Geetha	25	UB	$G_2P_1L_1$	Abruption-Grade-0	LSCS	2	4 / 10 7 / 10		+		-	UTI	
18.	Manju	27	UB	$G_2P_1L_1$	Abruption-Grade-3	VD	1.9	0	+			-	Shock	DIC
19.	Rani	26	Booked	$G_3P_2L_2$	Abruption-Grade-1	LSCS	1.8	3 / 10 7 / 10			+	-	BL TR	
20.	Parvin Banu	28	UB	$G_4P_3L_3$	Abruption-Grade-1	LSCS	2	4 / 10 7 / 10			+	-	UTI	CU
21.	Alagammal	25	Booked	Primi	Abruption-Grade-2	LSCS	1.9	0	+			-	BL TR / PPH	
22.	Sudha	23	UB	$G_3P_2L_2$	Abruption-Grade-1	LSCS	2	3 / 10 5 / 10			+	-	BL TR	
23.	Rajalakshmi	29	UB	$G_2P_1L_1$	Abruption-Grade-2	LSCS	2	5 / 10 7 / 10			+	-	BL TR	
24.	Indhumathi	25	Booked	$G_2P_1L_1$	Abruption-Grade-3	LSCS	1.7	0	+			-	Shock / PPH	DIC

25.	Vijaya	36	UB	$G_3P_2L_2$	Abruption-Grade-2	LSCS	1.9	5 / 10 7 / 10			+	-	BL TR	
26.	Pappathi	22	UB	Primi	Abruption-Grade-1	LSCS	2	3 / 10 5 / 10		+		-	BL TR	
27.	Amutha	27	UB	$G_3P_2L_2$	Abruption-Grade-1	VD	2	5 / 10 7 / 10	+			-	BL TR	
28.	Pichaimani	26	UB	$G_2P_1L_1$	Abruption-Grade-1	LSCS	2.1	5 / 10 7 / 10			+	-	BL TR	CU
29.	Rajathi	30	Booked	Primi	Abruption-Grade-3	LSCS	2.2	0	+			-	Shock	
30.	Valli	27	UB	$G_2P_1L_1$	Abruption-Grade-1	LSCS	3.1	6 / 10 8 / 10			+	-	BL TR	
31.	Palaniammal	25	UB	$G_3P_2L_1$	Abruption-Grade-2	LSCS	2.5	7 / 10 8 / 10			+	-	BL TR	
32.	Selvi	23	UB	Primi	Abruption-Grade-1	LSCS	2.4	3 / 10 5 / 10	+			-	WI	
33.	Rajalakshmi	26	Booked	$G_2P_1L_1$	Abruption-Grade-2	LSCS	2.2	5 / 10 7 / 10			+	-	BL TR	
34.	Sivaranjani	24	UB	$G_4P_3L_3$	Abruption-Grade-3	VD	2.3	0	+			-	Shock	CU
35.	Ananthi	24	UB	$G_2P_1L_1$	Abruption-Grade-1	LSCS	2.5	6 / 10 7 / 10			+	-	BL TR	
36.	Chitra	21	UB	Primi	Abruption-Grade-1	LSCS	2.4	6 / 10 7 / 10			+	-	BL TR	
37.	Shanthi	25	Booked	$G_2P_1L_0$	Abruption-Grade-1	LSCS	2.3	5 / 10 8 / 10			+	-	BL TR	

38.	Vanitha	24	UB	$G_3P_2L_2$	Abruption-Grade-3	LSCS	2.1	0	+			-	Shock	
39.	Maheswari	22	UB	Primi	Abruption-Grade-1	VD	2.1	3 / 10 5 / 10		+		-	WI / PPH	
40.	Arokiyameri	37	UB	$G_2P_1L_1$	Abruption-Grade-2	LSCS	2.3	6 / 10 8 / 10			+	-	BL TR	CU
41.	Suriya Begum	23	UB	Primi	Abruption-Grade-1	LSCS	2.2	7 / 10 8 / 10			+	-	BL TR	
42.	Mumthaj	29	UB	$G_4P_3L_3$	Abruption-Grade-2	LSCS	2.4	0	+			-	BL TR	
43.	Devi	32	UB	$G_3P_2L_2$	Abruption-Grade-1	VD	2.6	7 / 10 8 / 10			+	-	UTI	
44.	Mohana Lakshmi	27	Booked	$G_2P_1L_0$	Abruption-Grade-2	LSCS	2.8	5 / 10 8 / 10	+			-	BL TR / PPH	
45.	Kavitha	27	UB	$G_4P_3L_3$	Abruption-Grade-1	LSCS	2.9	6 / 10 7 / 10			+	-	BL TR	
46.	Banumathi	25	UB	$G_3P_2L_1$	Abruption-Grade-2	LSCS	3	0	+			-	BL TR	CU
47.	Adhi Lakshmi	31	UB	$G_2P_1L_0$	Abruption-Grade-2	LSCS	2.7	8 / 10 9 / 10			+	-	BL TR	
48.	Gandhimathi	27	UB	$G_4P_3L_1$	Abruption-Grade-1	LSCS	2.6	6 / 10 8 / 10			+	-	BL TR	FEB - MOR
49.	Johnsi Mary	25	UB	Primi	Abruption-Grade-2	LSCS	3.4	7 / 10 8 / 10	+			-	BL TR	
50.	Kaliammal	35	Booked	$G_2P_1L_1$	Abruption-Grade-1	LSCS	1.7	4 / 10 7 / 10			+	-	WI	

51.	Veeramani	20	UB	Primi	Abruption-Grade-2	LSCS	2.5	8 / 10 9 / 10			+	-	BL TR	
52.	Kalamani	30	UB	$G_3P_2L_2$	Abruption Grade-3	VD	2.7	0	+	-	-	+	DIC / ARF	
53.	Banupriya	19	UB	Primi	Abruption-Grade-2	LSCS	2.5	8 / 10 9 / 10			+	-	BL TR	
54.	Johthi	30	UB	$G_2P_1L_1$	Abruption Grade-3	LSCS	3	3 / 10 7 / 10		+		-	BL TR	
55.	Mariyayee	21	Booked	Primi	Abruption-Grade-1	LSCS	2.5	5 / 10 7 / 10			+	-	BL TR	FEB - MOR
56.	Lakshmi	35	UB	$G_3P_2L_2$	Abruption-Grade-2	VD	1.7	4 / 10 7 / 10	+			-	BL TR	
57.	Nargees Banu	27	UB	$G_2P_1L_1$	Abruption-Grade-1	LSCS	2.5	8 / 10 9 / 10			+	-	CU	
58.	Pitchammal	28	UB	$G_2P_1L_1$	Abruption-Grade-2	LSCS	2.7	0	+			-	BL TR	
59.	Selvi	26	UB	$G_3P_2L_2$	Abruption-Grade-1	LSCS	2.5	6 / 10 7 / 10			+	-	BL TR / PPH	
60.	Kousalya	24	Booked	$G_2P_1L_1$	Abruption Grade-3	LSCS	2.4	6 / 10 8 / 10			+	-	BL TR	
61.	Vennila	25	UB	$G_4P_3L_3$	Abruption-Grade-1	VD	3.3	7 / 10 8 / 10			+	-	CU	
62.	Ponarasi	25	UB	$G_3P_2L_2$	Abruption-Grade-2	LSCS	2.3	0	+			-	BL TR	
63.	Alamelu	23	UB	Primi	Abruption-Grade-2	LSCS	3.4	6 / 10 8 / 10			+	-	BL TR	CU

64.	Deeba	23	UB	Primi	Abruption-Grade-1	LSCS	2.1	6 / 10 7 / 10	+			-	BL TR	FEB - MOR
65.	Malarkodi	28	Booked	$G_2P_1L_1$	Abruption-Grade-2	LSCS	2.5	8 / 10 9 / 10			+	-	BL TR	
66.	Ramayee	25	UB	Primi	Abruption-Grade-1	VD	2.4	6 / 10 8 / 10			+	-	BL TR	
67.	Gomathi	30	UB	$G_4P_3L_3$	Abruption-Grade-2	LSCS	2.5	0	+			-	BL TR	
68.	Thanalakshmi	25	UB	$G_2P_1L_1$	Abruption-Grade-1	LSCS	3.4	6 / 10 8 / 10	+			-	BL TR	FEB - MOR
69.	Sathya Sheela Jecili	25	UB	Primi	Abruption-Grade-2	VD	2.6	7 / 10 8 / 10			+	-	BL TR	
70.	Lakshmi	25	Booked	Primi	Abruption-Grade-1	LSCS	2.8	6 / 10 8 / 10			+	-	BL TR	
71.	Malarkodi	33	UB	$G_3P_2L_2$	Abruption-Grade-2	VD	2.9	6 / 10 8 / 10	+			-	BL TR	
72.	Sumathi	20	UB	$G_2P_1L_1$	Abruption Grade-3	LSCS	3	7 / 10 8 / 10	+			-	BL TR	
73.	Sushila	28	UB	$G_2P_1L_1$	Abruption-Grade-1	LSCS	1.5	3 / 10 7 / 10			+	-	BL TR	FEB - MOR
74.	Pushpa	26	Booked	$G_3P_2L_2$	Abruption-Grade-2	LSCS	2.7	0	+			-	BL TR	
75.	Sundari	21	UB	Primi	Abruption-Grade-1	VD	3.4	8 / 10 9 / 10			+	-	BL TR	FEB - MOR
76.	Stella Mari	24	UB	$G_3P_2L_2$	Abruption-Grade-2	LSCS	2.7	0	+			-	BL TR	

77.	Santhana Mari	27	UB	$G_2P_1L_1$	Abruption Grade-3	LSCS	2.1	6 / 10 8 / 10	+			-	BL TR	
78.	Sudha	20	Booked	Primi	Abruption-Grade-1	VD	3.5	3 / 10 5 / 10		+		-	BL TR	FEB - MOR
79.	Haseena Begum	29	UB	$G_2P_1L_1$	Abruption-Grade-2	LSCS	2.9	8 / 10 9 / 10			+	-	BL TR	
80.	Pounuthai	23	UB	Primi	Abruption-Grade-1	VD	2.8	0	+			-	BL TR	
81.	Kodimalli	24	UB	Primi	Abruption-Grade-1	LSCS	2.7	8 / 10 9 / 10			+	-	BL TR	FEB - MOR
82.	Pappathi	30	Booked	$G_3P_2L_2$	Abruption-Grade-1	LSCS	2.6	6 / 10 8 / 10	+			-	PPH / CS-H	
83.	Mariammal	30	UB	$G_2P_1L_0$	Abruption-Grade-1	VD	2.6	8 / 10 9 / 10			+	-	BL TR	
84.	Deiva Gantham	27	UB	$G_2P_1L_1$	Abruption-Grade-3	LSCS	2.10	0	+			+	BL TR	
85.	Easwari	39	UB	$G_4P_3L_3$	Abruption-Grade-1	LSCS	2.80	8 / 10 9 / 10			+		BL TR	
86.	Malarkodi	28	UB	$G_4P_3L_3$	Abruption-Grade-0	LSCS	2.2	5 / 10 6 / 10		+			CU	
87.	Palaniammal	23	Booked	Primi	Abruption-Grade-1	VD	2.5	0	+				BL TR	
88.	Chandra	28	UB	$G_2P_1L_1$	Abruption-Grade-1	LSCS	2.8	8 / 10 9 / 10			+		BL TR	
89.	Chitra	25	UB	$G_3P_2L_2$	Abruption-Grade-0	VD	2.5	5 / 10 6 / 10	+				BL TR	

90.	Uma Maheswari	23	Booked	$G_4P_3L_2$	Abruption Grade-3	LSCS	2.9	7 / 10 8 / 10			+		BL TR / PPH	
91.	Ponnammal	26	UB	$G_3P_2L_2$	Abruption-Grade-1	LSCS	2.2	0	+				BL TR	
92.	Anjali Devi	27	UB	$G_4P_3L_3$	Abruption-Grade-1	LSCS	2.25	7 / 10 8 / 10			+		BL TR	
93.	Radhika	24	UB	Primi	Abruption-Grade-1	VD	2.6	0	+				WI	
94.	Rani	35	UB	$G_3P_2L_2$	Abruption-Grade-0	LSCS	3.0	7 / 10 8 / 10			+		UTI	
95.	Kala Mari	30	Booked	$G_2P_1L_1$	Abruption-Grade-1	LSCS	2.8	4 / 10 5 / 10		+			BL TR / PPH	
96.	Saraswathi	29	UB	$G_3P_2L_2$	Abruption-Grade-1	LSCS	2.5	0	+				CU	
97.	Thenmozhi	29	UB	$G_4P_3L_3$	Abruption-Grade-0	VD	2.9	8 / 10 9 / 10			+		BL TR	
98.	Easwari	23	UB	$G_2P_1L_1$	Abruption-Grade-1	LSCS	2.9	0	+				BL TR	
99.	Jeyanthi	23	UB	$G_4P_3L_2$	Abruption-Grade-0	LSCS	2.1	6 / 10 8 / 10		+			BL TR	
100.	Backiyam	27	Booked	$G_4P_3L_2$	Abruption-Grade-1	LSCS	2.7	0	+				BL TR	
101.	Panchavarnam	23	UB	Primi	Abruption-Grade-1	VD	2.0	6 / 10 8 / 10			+		UTI	
102.	Seethe	25	UB	$G_3P_2L_2$	Abruption-Grade-1	LSCS	2.1	0	+				BL TR	
103.	Parameswari	25	UB	$G_2P_1L_1$	Abruption-Grade-0	LSCS	2.5	0	+				BL TR / PPH	.

ABBREVIATIONS

UB	-	Unbooked
FEB MOR	-	Febrile Morbidity
CU	-	Couvelaire Uterus
ARF	-	Acute Renal Failure
BL TR	-	Blood Transfusion
WI	-	Wound Infection
PPH	-	Post Partum Hemorrhage
UTI	-	Urinary Tract Infection
DIC	-	Disseminated Intravascular Coagulation
VD	-	Vaginal Delivery
CS / H	-	Caesarian Hysterectomy